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U.S. Environmental Protection Agency
Region 10

Baseline Human Health And Ecological Risk Assessments For Monsanto Chemical Corporation Superfund Site Soda Springs, Idaho

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BASELINE
HUMAN HEALTH AND ECOLOGICAL
RISK ASSESSMENTS

for the

MONSANTO CHEMICAL CORPORATION
ELEMENTAL PHOSPHORUS FACILITY SUPERFUND SITE
SODA SPRINGS, IDAHO

Prepared for:

U.S. Environmental Protection Agency
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ACRONYMS

1E-03	Example of scientific notation ($1x10^{-3}$) used in this document.
AVG	Refers to Average Exposure - See RME.
CERCLA	Comprehensive Environmental Response, Compensation, and Liability Act
COC	Chemical of Concern. A chemical associated with a Site which would cause a hazard or risk, given the modelling assumptions of the assessment.
COPC	Chemical of Potential Concern. A chemical found to be associated with a Site which may potentially threaten human or environmental health.
EPA	U.S. Environmental Protection Agency (USEPA)
EPC	Exposure Point Concentration. The chemical concentration in a specific media (soil, water, air, etc.) to which a biological receptor is exposed.
ERA	Ecological Risk Assessment
HEAST	Health Effects Assessment Summary Tables
HI	The sum of HQs which have like toxicological endpoints. Like endpoints may include kidney damage, blood disease, etc.
HQ	Hazard Quotient. A dimensionless quotient of chemical intake to RfD.
IRIS	Integrated Risk Information System
MCC	Monsanto Chemical Company
MCL	Maximum Contaminant Level in water (Clean Water Act). SMCL = secondary criteria, MCLG = goal.
NRC	Nuclear Regulatory Commission
PSCSR	Preliminary Site Characterization Summary Report
RAGS	Risk Assessment Guidance
RBC	Risk Based Concentration. Given a set of exposure assumptions, the concentration in a particular media which would cause a hazard or risk (e.g., HQ > 1, or a risk > 1E-06).
RESRAD	A Department of Energy computer program used to determine exposure and risks to Radionuclides and Radiation.
RfD	Reference Dose. A dose which would not be expected to result in adverse toxic effects in a sensitive individual.
RI/FS	Remedial Investigation/Feasibility Study
RMC	Reasonable Maximum Concentration. An EPC normally characterized by the UCL of the data set.
RME	Reasonable Maximum Exposure. The maximum quantity of media that an individual would be exposed to over a lifetime. This value is maximized by maximizing assumptions regarding exposure frequency, exposure duration, intake (of media), etc.
SF	Slope Factor. The relationship between chemical or radionuclide exposure, and the probability of developing cancer as a result of that exposure.
TW	One of Monsanto's prefixes for well-identifiers.
UCL	Upper Confidence Limit of the mean value of a set of a population. Normally viewed as the 95% probability that the true mean of that population would fall below that value.
UTL	Upper Tolerance Limit. The upper 95th percent confidence limit on the 95th percentile of a data set.

Remedial Investigation/Feasibility Study

In general, the Remedial Investigation/Feasibility Study is a process designed to characterize a site, assess the nature and extent of contamination at a site, evaluate the potential risk to human health and the environment and develop and evaluate remedial alternatives. An RI/FS has two primary objectives:

> To provide information to assess the risks posed to public health and the environment by the site;
>
> To evaluate a range of remedial alternatives (treatment controls, institutional controls, or a combination of these plus a no action alternative) to reduce the risks found and/or exposure to them, based on site specific criteria.

The process is composed of a Remedial Investigation, which includes the actual field data collection and risk assessment process (corresponding to the first objective), and the Feasibility Study, which develops and evaluates remedial alternatives. The Remedial Investigation and Feasibility Study include iterative activities and will overlap in timing. Data collected in the Remedial Investigation influences the development of remedial alternatives in the Feasibility Study, which in turn affects data needs and the scope of treatability studies, if any are necessary. The distinction between the two phases is made to emphasize the focus of the studies.

The specific tasks required to perform an RI/FS may vary, and are phased in accordance with a site's complexity and the amount of available information. A phased RI/FS process was used at the Monsanto site to facilitate early identification of data collection requirements. These requirements are intended to characterize the site by effectively describing contaminant concentration, fate and transport, and exposure pathways so that sufficient information is available to determine risk and if necessary, evaluate and compare the remedial alternatives.

The purpose of the baseline risk assessment at a Superfund site is to characterize the current and potential risk that the site poses to human health and the environment. The results of the baseline risk assessment may indicate that the site poses substantial, little, or no threat, and that substantial, limited, or no further response activity is required. If the baseline risk assessment indicates that action may be necessary, remediation goals are determined for the site contaminants and a Feasibility Study is performed to assemble alternatives and evaluation of how each viable alternative addresses the risks characterized in the risk assessment.

HUMAN HEALTH RISK ASSESSMENT - EXECUTIVE SUMMARY
Monsanto Superfund Site
Soda Springs, Idaho

The Monsanto facility in Soda Springs was placed on the National Priorities List of Superfund Sites in 1990. Sites placed on the Superfund National Priorities List must go through an environmental investigation to determine the nature and extent of contamination and the associated risks, followed by a feasibility study to evaluate alternatives to reduce or eliminate risks associated with the contamination.

This document presents an assessment of the potential risks associated with the Monsanto Elemental Phosphorus Plant in Soda Springs, Idaho. It has been prepared by the U.S. Environmental Protection Agency (EPA) as part of a detailed environmental investigation of the Monsanto facility, known as a Remedial Investigation and Feasibility Study. The rest of the Remedial Investigation is being performed by Monsanto in accordance with the requirements of the Superfund law, under EPA and State oversight. This executive summary is specifically written for the lay reader who may have a less technical background but an interest in the findings of the risk assessment.

Please note that the rest of this risk assessment is primarily written for the technical reader who is familiar with human health risk assessment and the references cited. It is not a completely stand-alone document, although every effort has been made to provide information about assumptions, equations, methods and values used, at least by reference. The baseline Human Health and Ecological Risk Assessments (see part two for the Ecological) will eventually be incorporated into the Remedial Investigation Report being prepared by Monsanto. That Report will include the detailed information summarized and/or referred to in this document.

Findings of the Remedial Investigation So Far

The production of elemental phosphorus at the Monsanto facility creates several byproducts and wastes containing heavy metals and radionuclides. Constituents from these materials have been found in the ground water beneath the plant and in soils near the facility at levels above background or naturally occurring levels. In groundwater, the chemicals of concern include: cadmium, fluoride, and selenium. In soils and in source piles, the chemicals of concern include metals (chiefly arsenic, beryllium and cadmium) and radioactive substances (i.e., radionuclides).

The extent of contamination at levels of concern to human health appears to be limited to the facility itself, soils immediately surrounding the plant site, and groundwater beneath and immediately downgradient from the facility. The ground water is apparently contaminated due to infiltration resulting from historic use of unlined ponds for on-site disposal of waste materials. Sediments at some locations in Soda Creek downstream from the facility contain

chemicals of concern but at levels below those which pose risks to humans (potential ecological risks from the sediments are still being evaluated). Windblown dust and other airborne emissions cause contaminants to leave the site and reach surrounding soils.

Purpose of this Risk Assessment

The purpose of this baseline risk assessment is to provide an evaluation of risks to human health from potential releases of hazardous substances at or from the Monsanto site in the absence of any further cleanup action. This information is required to help determine what cleanup actions, if any, may be necessary to reduce risks.

At Superfund sites which are operating facilities, such as the Monsanto facility, EPA and the Occupational Health and Safety Administration (OSHA) have complementary statutory authorities to ensure the safety and health of the workforce, through implementation of the Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA) and the Occupational Safety and Hygiene (OSH) Act. This risk assessment has attempted to address only the CERCLA mandate to characterize the current and potential risks posed to human health through uncontrolled releases of hazardous substances to the environment and to support evaluation of the need for cleanup or other actions to reduce exposure. Potential risks that are not attributable to uncontrolled releases to the environment (i.e., exposure to high temperatures, noise or controlled emissions inside the furnace buildings) are beyond the scope of this assessment. If EPA identifies safety concerns within the area of OSHA responsibility, those concerns will be discussed with or referred to OSHA, as appropriate.

Slag disposed of at the facility has been evaluated in this assessment. Slag used for other purposes away from the facility, such as in roads and homes, is being evaluated as part of a different study and is not included in this assessment.

What is Meant by the Term "Risk"?

In reading this document it is important to keep in mind the difference between risk and toxicity, (or hazard, as in hazardous substances). In risk assessments, risk is a function of exposure and toxicity. Highly toxic substance may pose no risk if there is no way for anyone to be exposed to them. This document assesses the potential risks to people from exposure to hazardous substances at or from the Monsanto facility, if no further action is taken to reduce potential risks.

Summary Findings and Conclusions

No acute (immediate, short-term) risks were identified. The Soda Springs City drinking water supplies are unaffected by the site.

Long-term risks were calculated for four different scenarios: 1) current residential, 2) future residential, 3) current industrial and 4) future industrial. The current residential scenario assesses potential risks to actual residents currently living in the general vicinity of the Monsanto facility. The future residential scenario assesses potential risks in areas immediately adjacent to the facility which could potentially be occupied at a future date. Similarly, the current industrial scenario calculates potential risks to current workers within the boundaries of the Monsanto facility; while the future scenario assesses potential risks to employees of industrial operations which could potentially occupy the site if and when the Monsanto facility closes. For a more thorough description of these scenarios and the assumptions used, please refer to Section 3.0 of this assessment.

Federal environmental laws and regulations recognize that estimates of very small risk levels are insignificant. The concept of *de minimus* risk refers to a specific level below which risks are so small that they are not of concern. In risk assessments, government agencies recognize that cancer risks less than 1-in-1,000,000 are generally *de minimus* and risks between 1-in-1,000,000 and 1-in-10,000 are within the generally acceptable range. Risks greater than 1-in-10,000 are generally regarded as the "point of departure"; site cleanup is generally required at this level. The EPA Superfund program has adopted these regulatory ranges, which are used to place the estimated excess lifetime cancer risks into context (EPA 1992d).

For evaluating noncarcinogenic effects, EPA defines acceptable exposure levels as those to which the human population, including sensitive subgroups such as children, may be exposed without adverse effects during a lifetime or part of a lifetime, incorporating an adequate margin of safety (EPA 1989a). This acceptable exposure level is approximated best by a Hazard Index (HI) equal to 1. If the HI is less than 1, adverse effects usually would not be expected. However, adverse effects may occur when the HI exceeds 1.

Risks are characterized separately for cancer (carcinogenic) and non-cancer effects. Carcinogenic risks from chemicals were also evaluated separately from carcinogenic radionuclides.

Exposure to chemicals of concern via **pathways** resulting in cancer risks greater than 1-in-1,000,000, or a non-cancer hazard quotient greater than 1 (the level below which no adverse effects are expected), were identified as concerns. The specific chemicals contributing to those risks are identified as Chemicals of Concern. The chapter discusses in detail the exposure pathways, chemicals of concern and risks associated with each scenario.

Several long-term <u>potential</u> risks to residents in the vicinity of the facility and to public health within the facility were identified. The risk estimates for these scenarios, discussed briefly below, are of sufficient magnitude based on EPA guidance and CERCLA [Superfund] requirements to warrant evaluation of alternatives to determine what, if any,

remedial action could be taken to reduce risks (a Feasibility Study). Whether cleanup is necessary, and if so what action(s) are needed, will be decided after that study and a public comment period.

Current Residential Scenario

For the purposes of evaluating potential risks posed to current off-site residents, it was assumed that individuals would be residing in the same location for 30 years. The risks associated with the current residential scenario are summarized below:

- Current residents in the immediate vicinity of the site are potentially exposed to chemical and radionuclide contaminants originating from the Monsanto facility. The potentially affected area is presented in Figure 1-3.

- The upper-bound estimate of incremental lifetime cancer risks from ingestion of chemical contaminants (mainly arsenic, beryllium, and cadmium) is within EPA's acceptable range (i.e., ranging from below background to 4-in-100,000).

- The upper-bound estimate of incremental lifetime cancer risks from ingestion of radionuclide contaminants is below background at all locations.

Future Residential Scenario

For the purposes of evaluating potential risks posed to future residents, it was assumed that individuals would be residing at the same location for 30 years. To be conservative, the possibility of former residences along the southern fenceline (now owned by Monsanto) being reoccupied was considered. The risks associated with the future residential scenarios are summarized below:

- The upper-bound estimate of incremental lifetime cancer risks from ingestion of chemical contaminants (mainly arsenic, beryllium, and cadmium) are within EPA's acceptable range (i.e., from below background to 1-in-10,000).

- The upper-bound estimate of incremental lifetime cancer risks from ingestion of radionuclide contaminants ranges from below background to above EPA's point of departure, (i.e., 1-in-1,000) at the southern fenceline (due to radium-226 and its decay products).

- Future residents living south of the facility could be exposed to additional contaminants if they were to install wells and use groundwater as a source of drinking water. Consumption of groundwater containing cadmium, fluoride, and selenium would yield a noncancer risk estimate that marginally exceeds an HI of one.

Current Industrial Scenario

Potential exposures to site-related contaminants under current working conditions were derived using time-and-motion data provided by Monsanto Chemical Corporation. Consequently, variables such as the daily exposure time, the number of days per year on-site, and the degree to which mechanical equipment provided shielding from radioactive emissions varies from area-to-area.

- Current ingestion and inhalation exposures to chemical carcinogens (arsenic, beryllium, and cadmium) yield incremental lifetime cancer risks within EPA's acceptable range (i.e., ranging from less than 1-in-1,000,000 at the Treater Dust area to 3-in-100,000 at the Underflow Solids area).

- Current ingestion and inhalation of radionuclides in addition to external exposure (principally from radium-226 and its decay products) yield incremental lifetime cancer risks ranging from 7-in-100,000 at the Treater Dust area to 5-in-10,000 at the Baghouse Dust and Slag Areas. Risks are approximately an order of magnitude higher than background.

Future Industrial Scenario

Future industrial activities at the site were evaluated assuming that the Monsanto facility was no longer operational and that the contaminated materials present on-site were not remediated prior to closure. It was assumed that all workers were present on-site for 8 hours/day, 250 days/year, for 25 years and are unshielded from radioactive contamination. [Note that Monsanto has assured EPA that the company will not abandon this facility without taking appropriate actions to ensure public safety.]

- Future ingestion and inhalation exposures to chemical carcinogens (arsenic, beryllium, and cadmium) yield incremental lifetime cancer risks within EPA's acceptable range (i.e., ranging from 1-in-100,000 at the Treater Dust and Slag areas to 6-in-100,000 at the Underflow Solids area).

- Future ingestion and inhalation of radionuclides in addition to external exposure (principally from radium-226 and its decay products) yield incremental lifetime cancer risks in excess of EPA's acceptable range, (i.e., 1-in-1,000 at all areas except the Nodules and Slag areas, where the risks were 2-in-1,000). Risks were approximately an order of magnitude higher than background.

General Uncertainties in the CERCLA Risk Assessments:

While sufficient information was provided to perform the baseline human health and ecological risk assessments, some issues were identified that could not be fully evaluated with the available information. For human health, the estimated risks from inhalation of dusts are considered to have a high degree of uncertainty, because of the reliance on modelled data rather than actual sample results (in areas where air monitoring has not been conducted). Additional uncertainty is associated with the gamma radiation measurements obtained during the remedial investigation, due to the instrumentation used, the estimates of the cancer risks associated with external exposure are conservative. These issues will be further discussed with Monsanto to determine the available options to reduce the uncertainty, if necessary. For a more detailed description of the uncertainties associated with this assessment, please refer to Section 5.0 of this assessment.

Next Steps

EPA, the State, and Monsanto will continue to work together to develop the necessary information to make appropriate decisions for this site. This document (including the Ecological Risk Assessment which follows) is being provided to Monsanto for use as they complete the Draft Remedial Investigation Report, and to the public for information purposes.

After the Remedial Investigation Report is completed, Monsanto will submit draft site remediation (cleanup) goals to EPA and the State for review. The draft goals will be used by Monsanto as they proceed to evaluate potential actions to reduce risk in what is known as a Feasibility Study.

This risk assessment and all other documents produced during the RI/FS process are draft documents, subject to new information developed later in the RI/FS and to a public comment period before decisions are made. Public input, comments and questions are always encouraged and should be addressed to: U.S. EPA Superfund (HW-113), 1200 Sixth Avenue, Seattle, Washington 98101. A public meeting will be held to discuss this document and the RI Report after the draft remediation goals are submitted to EPA by Monsanto.

1.0 INTRODUCTION

This baseline risk assessment is part of the Remedial Investigation/Feasibility Study (RI/FS) of the Monsanto Chemical Company (MCC) Soda Springs Elemental Phosphorus Plant. Because the RI/FS is an analytical process designed to support risk management decision-making for Superfund sites, the assessment of health and environmental risk plays an essential role in the RI/FS.

The primary objective of this baseline risk assessment is to evaluate potential chemical hazards and carcinogenic risks to human health that are attributable to the MCC Plant in the absence of any remedial action. Actual and potential hazards to local residents and workers that are attributable to chemicals released to the environment from the Plant are evaluated in this document. Full documentation of the nature and extent of contamination, as well as background characterization is presented in the RI report and is not presented in this document.

1.1 OVERVIEW

The elemental phosphorous production facility in Soda Springs, Idaho, has been operated by the Monsanto Chemical Company since the mid-1950s. Prior to Monsanto's purchase of the property in 1952, the site was used for agricultural and domestic purposes (Golder 1992). Figure 1-1 provides the location of the site.

The production of elemental phosphorous at the MCC Plant uses a thermal process which treats the phosphate ore in electric-arc furnaces. This industrial process creates several byproducts (mainly heavy metals and radionuclides) which have accumulated in various environmental media. Approximate areas of ground water and soil contamination above background are illustrated in Figures 1-2 and 1-3, respectively. This baseline human health risk assessment was undertaken in order to assess the human health hazards and risks to potentially-exposed populations near the Plant in the event no further action is taken to reduce site-related risks.

Previous investigations at the MCC Plant have focused mainly on ground water quality. Monsanto installed several ground water monitoring wells prior to 1984. Golder Associates, the primary contractor for MCC, conducted a hydrogeological and surface water investigation in 1984. In 1988, a Site Inspection (SI) was undertaken at the request of the U.S. Environmental Protection Agency (EPA). The SI report submitted to the EPA concluded that on-site waste ponds contained toxic chemicals, and that ground water in the vicinity also contained chemicals released from the Monsanto Plant. Currently, 46 monitoring wells and several springs and production wells are being used to monitor ground water quality.

The MCC Plant was placed on the Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA) National Priority List in August 1990, primarily because of potential effects on ground water quality from operational practices. Monsanto began environmental sampling to determine the nature and extent of contamination subsequent to an Administrative

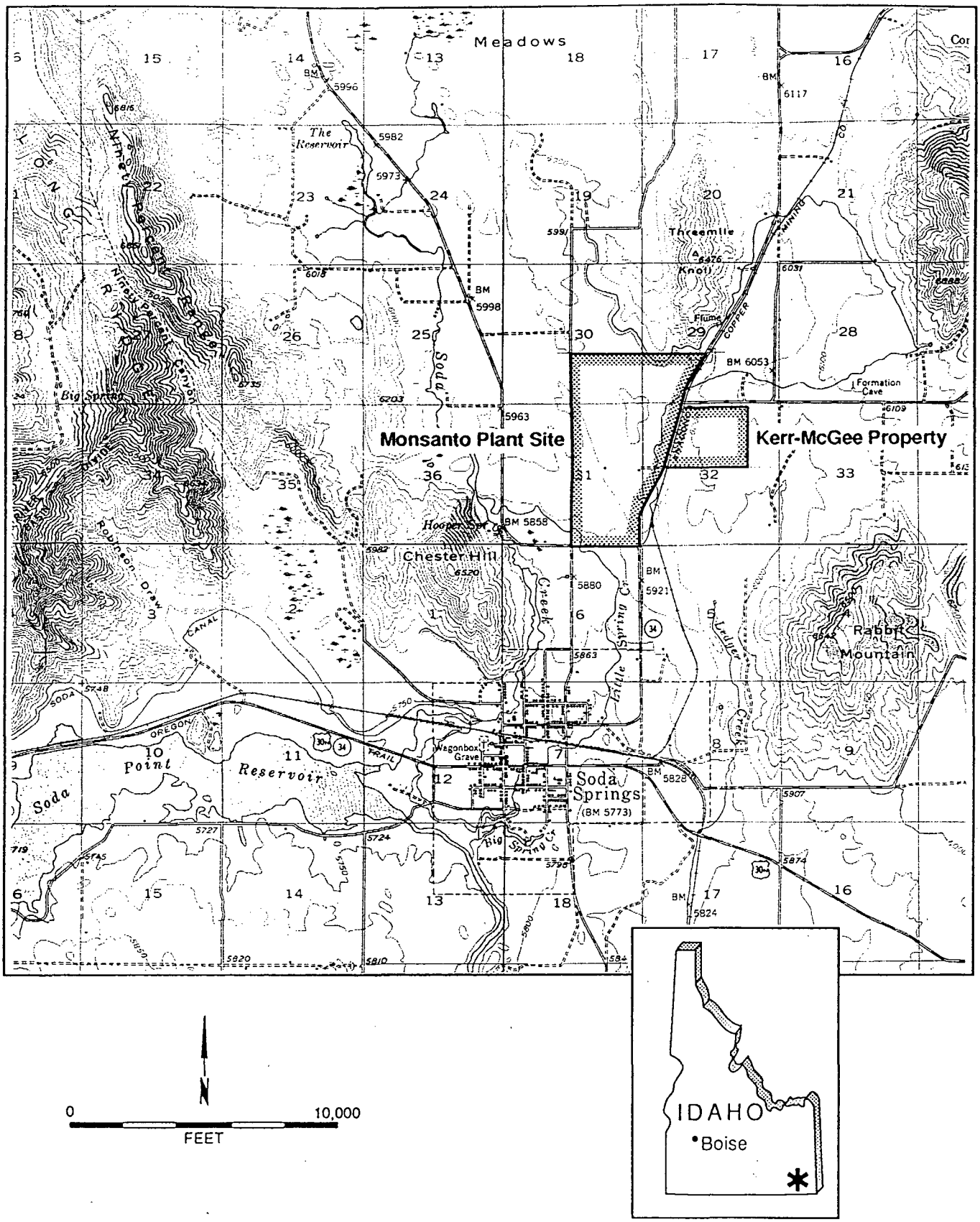

FIGURE **1-1**

LOCATION MAP

MONSANTO/PHASE 1 RI REPORT/ID

SOURCE: Topographic map of the USGS Soda Springs Quadrangle (1:62,500) 1948.

PROJECT NO. 913-1101.212 DRAWING NO. 27592 DATE 1/31/92 DRAWN BY EA

Golder Associates

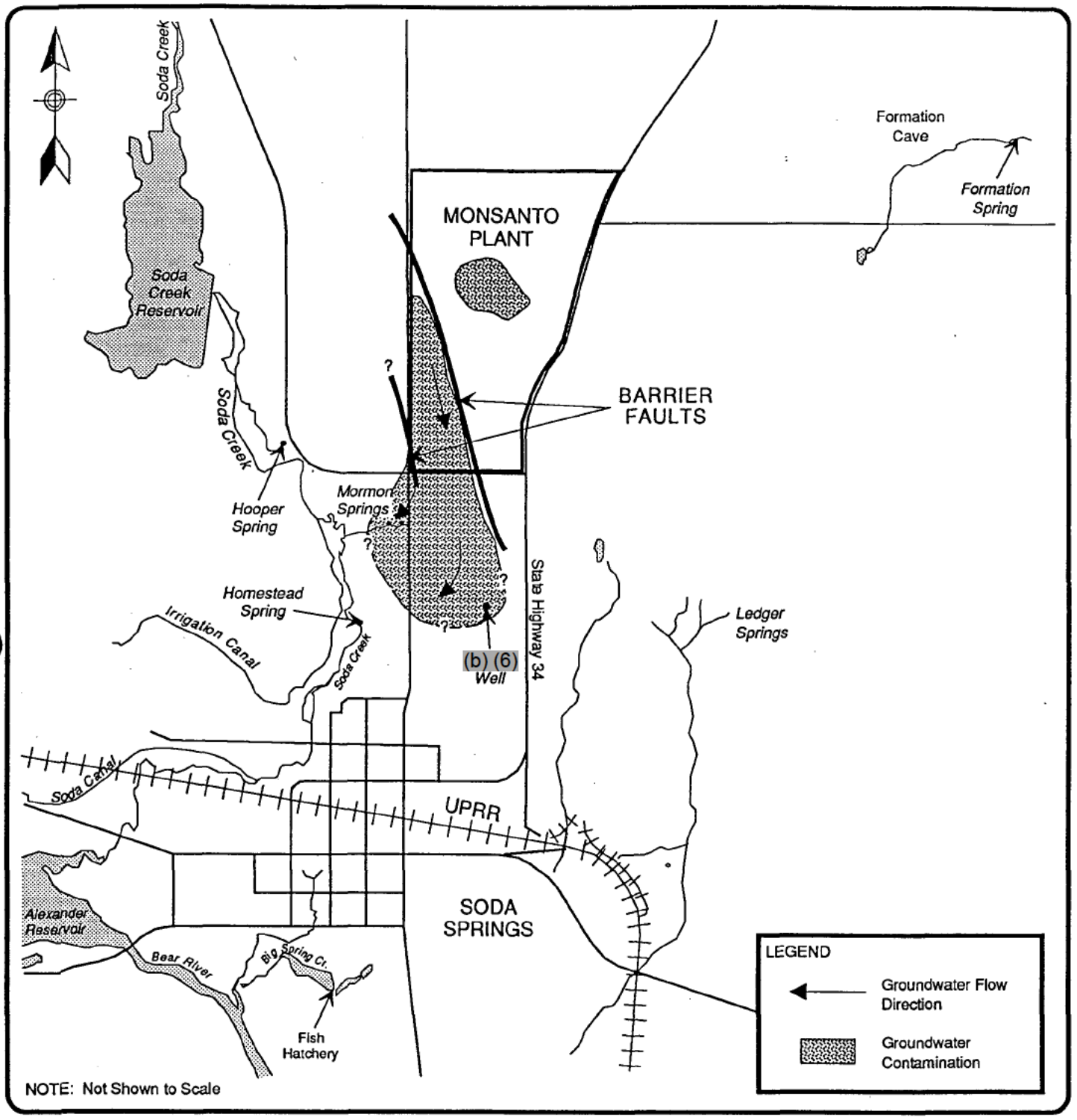

Figure 1-2

APPROXIMATE AREAS OF GROUNDWATER CONTAMINATION

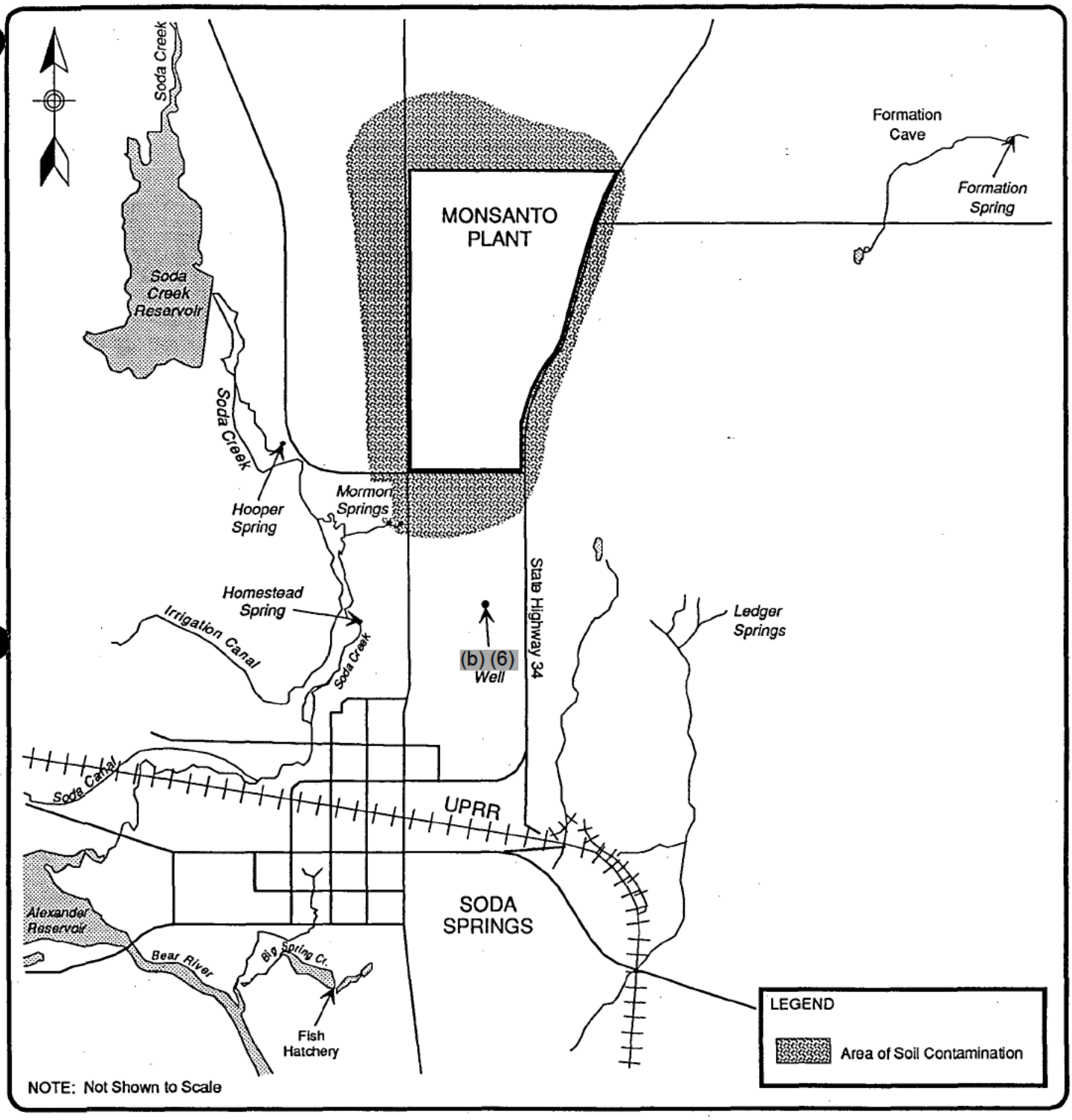

Figure 1-3

APPROXIMATE AREA OF SOIL CONTAMINATION
ABOVE BACKGROUND

Order on Consent for Remedial Investigation/Feasibility Study (RI/FS) issued to Monsanto in March 1991 by EPA. Phase I data were presented in the Preliminary Site Characterization Summary Report, submitted in April 1992. Several other reports and technical memoranda regarding Phase II data have been submitted. Relevant RI data have been used in this risk assessment.

1.2 SITE DESCRIPTION

The MCC Plant is located approximately 1 mile north of Soda Springs, Idaho (Figure 1-1). Monsanto owns approximately 540 acres at this location. Portions of the Monsanto property to the south and southwest of the Plant are agricultural. Rangeland and agricultural land predominate the area immediately surrounding the Plant.

Several retention ponds exist onsite. The effluent settling pond and the sewage lagoon are unlined. The settling pond is used to remove solids from the non-contact cooling water. The water is then discharged into Soda Creek under a National Pollution Discharge Elimination System (NPDES) permit. The currently-used lined ponds include the phossy water and seal water ponds. Other on-site sources of potential contaminants include the following: (1) a variety of stockpiled waste materials; (2) stack emissions; and (3) fugitive dusts. A complete description of the elemental phosphorus production process is found in the Preliminary Site Characterization Summary Report (Golder 1992).

1.3 SURFACE FEATURES AND LAND USE

The Monsanto Plant is located in the Bear River Basin, which is characterized by broad, flat valleys with a few scattered topographic features including cinder cones, rhyolitic domes, and uplifted fault blocks. The Plant lies at an elevation of approximately 6,000 feet in a tributary valley to the basin. Northwest trending mountain ranges, 8,000 feet in elevation, border the valley to the west. These mountains include the Chesterfield Range and the Soda Springs Hills. The Aspen Range lies approximately seven miles to the east of the Site. The northern boundary of the tributary valley is formed by the Blackfoot Reservoir, located approximately 13 miles north of the Site. Surface drainage in the valley, south of Blackfoot Reservoir, is predominantly to the south toward Alexander Reservoir.

Natural springs are important hydrologic features of the Bear River Basin. Soda Springs obtains its municipal water supply from Formation Spring and Ledger Spring. Formation Spring is located hydraulically upgradient, approximately 2.5 miles east-northeast of the MCC Plant. The Ledger Spring complex, also hydraulically upgradient, includes several springs located approximately 1.5 miles southeast of the Site.

The Site is located in an area characterized mostly by dry land and sagebrush-grass vegetal cover type, which occurs at the 5,000- to 7,000-foot elevation (U.S. Departments of Interior and Agriculture 1976). Although agricultural-type cover now dominates the surrounding area, most of the agricultural lands were once sagebrush-grass, dry lands, and riparian zones. The most common crops near the Plant are "small grains" (wheat and barley), some grasses, and alfalfa.

1.4 SCOPE OF THE BASELINE HUMAN HEALTH RISK ASSESSMENT

The scope of this assessment includes all potential chemical hazards and carcinogenic risks to human health that are attributable to uncontrolled releases of hazardous substances to the environment at or from the MCC Plant in the absence of any remedial action. Potential risks to human health in residential and industrial settings were evaluated under both current and future scenarios.

This assessment evaluates potential exposure associated with disposal of slag at the facility. However, the use of slag in the community for roads, etc., is the subject of a separate study and is beyond the scope of this assessment.

Workplace exposures or risks that are not attributable to uncontrolled releases to the environment (e.g., exposure to high temperatures, noise or controlled emissions inside the furnace buildings) are beyond the scope of this assessment. Such exposures are the purview of the Occupational Safety and Health Administration (OSHA).

EPA and OSHA have statutory responsibilities to ensure the safety and health of the public and America's workforce through the timely and effective implementation of a number of federal laws and implementing regulations. In some areas, the responsibilities of the agencies are separate and distinct. In others, they are complementary. EPA's responsibilities include the protection of public health and the environment by assuring compliance with federal environmental statutes and regulations. OSHA is responsible for enforcing the OSH Act, which has as its goal to assure, as far as possible, that every working man and woman has safe and healthful working conditions. OSHA standards provide limitations to the levels of hazardous materials to which workers may be exposed in their working day. The purpose of this risk assessment is to identify potential health threats that warrant consideration of potential CERCLA remedial action. If EPA identifies safety concerns within the area of OSHA responsibility, those concerns will be discussed with or referred to OSHA, as appropriate.

The Kerr-McGee Chemical Company operates a facility across the highway from the Monsanto site. Although the Kerr-McGee facility is also a Superfund site, this facility is beyond the scope of this assessment. Separate RI/FS's are being prepared for each site; information from both sites was considered in this risk assessment.

This risk assessment has been prepared by EPA using information gathered by Monsanto for the RI/FS. Risk assessment data needs were identified in the initial planning for the RI and have been refined as additional site characterization has been performed. All environmental samples collected and analyzed in the RI were evaluated for the risk assessment. Sufficient data were available to perform this risk assessment, although some data gaps have been identified which may need to be addressed before the RI/FS can be considered complete.

Although the RI/FS process and related risk information activities are often presented in a fashion that makes the steps appear sequential and distinct, in practice the process is highly interactive. This baseline human health risk assessment is the first part of the overall human health evaluation of the MCC facility. The other parts of the human health evaluation (the refinement of preliminary remediation goals and the remedial alternatives risk evaluation) will

be performed later in conjunction with the FS. Once complete, the RI/FS will provide decision-makers with a technical evaluation of the health threats posed at a site, a characterization of the potential routes of exposure, an assessment of remedial alternatives, and an analysis of the trade-offs in selecting one over the other.

The level of effort required to conduct a baseline risk assessment depends largely on the complexity of the site. In this case, the risk assessment has been complicated by several factors, including:

- The fact that this is an operating industrial facility, and is likely to remain as such;
- The presence of radionuclide as well as chemical health hazards (radionuclide hazards have traditionally been measured and evaluated differently than chemical hazards);
- Relatively high levels of background radioactivity in the area;
- The complexity of the underlying hydrogeology, reflecting the presence of fractured basalt and multiple ground water sources; and,
- The proximity to the Kerr-McGee facility.

The site specific objectives of this risk assessment include:

- Evaluation of data and chemicals of **potential** concern, which are then narrowed to a list of **chemicals of concern** discussed in chapters five and six of this document;
- Identification of potential human receptors and exposure pathways;
- Quantification of exposure;
- Characterization of human health risks to current and future receptors; and,
- Identification of data gaps that may require additional investigation.

1.5 APPROACH AND KEY ASSUMPTIONS

This human health assessment was conducted using appropriate EPA and Superfund guidance (USEPA 1989a; USEPA 1991a,b; USEPA 1992a,c).

Both current and future scenarios were developed to evaluate potentially significant human health risks. Total hazards and risks were calculated by analyzing scenarios based on multiple exposures within localized areas. All environmental samples collected and analyzed in the RI were evaluated for useability in this risk assessment, based on the scenarios selected.

Equations to assess chemical intake and associated risks, along with appropriate default parameters, were derived from EPA guidance documents. These exposure parameters yield conservative (i.e. health-protective) risk estimates. Key assumptions made before completing the risk assessment include:

- Chemical concentrations in environmental media and resulting exposures remain constant over time;
- Ground water could be used in a future residential scenario for drinking and household use; and,
- Except where site-specific exposure information has been documented, EPA default parameters are representative of the potentially exposed populations.

1.6 DOCUMENT ORGANIZATION

The remainder of the document is organized as follows:

Section 2.0 - Identification of Chemicals of Potential Concern. Site data relevant to the risk assessment are compared with background concentrations, as well as to toxicological reference values, to derive a list of chemicals for evaluation in the risk assessment.

Section 3.0 - Exposure Assessment. Exposure scenarios are developed that represent onsite and offsite current and future exposures. Intake and exposure factors are developed for each of these scenarios and are used to calculate hazards and risks.

Section 4.0 - Toxicity Assessment. The toxicological properties of site-related chemicals are discussed. Chemical-specific toxicity values used to predict hazards and risks are presented. Compounds that lack toxicity values are also discussed. Uncertainty factors associated with reference doses are presented in this section; a full list of specific reference doses and slope factors is included in Appendix A. Toxicological profiles are presented in Appendix D.

Section 5.0 - Risk Characterization. In this section, hazard and risk calculations are presented, based on information presented in the exposure and toxicity assessments. Hazard and risk calculations are presented in Appendix B. The uncertainties associated with exposure and toxicological parameters are discussed. Risks at background are presented, and the calculated hazards and risks are evaluated.

Section 6.0 - Summary and Conclusions. The results of the human health assessment are briefly discussed.

Section 7.0 - References. The references used to prepare this report are listed.

2.0 IDENTIFICATION OF CHEMICALS OF POTENTIAL CONCERN

This section evaluates data collected during the Remedial Investigation (RI) of the Monsanto Chemical Company Site. RI sampling events have been ongoing since the fall of 1991. Data collected through the fall of 1993 have been validated and are evaluated for usability in this section. Chemicals of potential concern (COPCs) are identified based on comparisons to background levels and risk-based screening criteria.

2.1 DATA EVALUATION

RI data from each sampling round were reviewed for data quality and usability. Details of the RI sampling protocols, analytical methods, quality assurance records, and results for the various media are presented in several RI reports (Golder 1992, 1993a,b,c,d,e). Data quality objectives (DQOs) were developed during various stages of RI sampling plans. DQOs were reviewed by EPA throughout the RI process to ensure that data would be of sufficient quality for assessment of potential human health risks. Data gaps were also identified. The detection limits for all chemicals were evaluated and deemed appropriate for this risk assessment. Error margins associated with radionuclides were evaluated for potential data uncertainties. Chemicals not detected in any media were excluded from the risk assessment.

In the calculation of exposure point concentrations (EPCs), all non-detected values were assigned a value equivalent to one half the sample quantitation limit, provided the analyte was detected in at least one other sample.

Unfiltered water samples were used in this risk assessment because potential drinking water may not be filtered. Equipment blanks, blind duplicate samples, and matrix spike samples were analyzed as indicators of laboratory and sampling precision, and were not used to calculate hazards or risks in this assessment.

Chemical data were also available from Soda Creek sediments and several springs located south of the MCC Plant. These data were not used in the human health assessment because human ingestion of sediments or spring water within the Soda Creek drainage is unlikely to occur.

Media and samples used in the assessment, as indicated by the conceptual site model, included offsite ground water, offsite soil, onsite source materials, and air. Unlined industrial ponds have contaminated areas of ground water. Onsite workers may be directly exposed to the large onsite waste piles; blowing dust from these piles, in addition to materials originating from the stacks, may affect air quality. Airborne contaminants may deposit onto offsite soils and cause contamination of that medium. Documentation of these processes, as well as RI data used in this risk assessment, is presented in the following documents:

- Air Dispersion Modelling Report (SENES 1993) and subsequent data (SENES 1994a,b)
- Preliminary Site Characterization Summary Report (Golder 1992)

- Vadose zone and Aquifer Material Source Evaluation (Golder 1993a)
- Hydrogeologic Investigation Memorandum (Golder 1993b)
- Fate and Transport Modelling Memorandum (Golder 1993c)
- Ground water Quality Memorandum (Golder 1993d)
- Sediment and Soils Investigations Memorandum (Golder 1993e)

Inorganic and radionuclide chemical data were screened in the media described above and used in the exposure assessment (Section 3.0). The screening criteria included background and risk-based concentrations (RBCs). Total gamma radiation in onsite sources and offsite soils was also evaluated.

Analysis was also performed for radionuclides because industrial processes indicated that uranium-238 and its decay products would be present in plant waste streams. Various isotopes of this chain were sampled in order to provide a thorough assessment of radionuclides associated with the Plant. Specific radionuclide decay products, represented by "+D", were considered in this risk assessment. This risk assessment used the slope factor methodology for assessing incremental risk from these radionuclides.

Evaluation of incremental risks from gamma radiation exposure was also performed. This approach follows a different methodology from that employed for specific radionuclides. During the Monsanto RI, gamma readings were collected for the purpose of identifying areas at which radionuclide concentrations were elevated with respect to background soil levels. The measured gamma field includes radiation from radionuclides measured as part of the RI, radionuclides not measured as part of the RI, and cosmic gamma radiation. As an addendum to this risk assessment, gamma risks were evaluated for the purpose of comparing the methodology presented in the main text with the alternative methodology presented in Appendix E.

Organic chemicals were not evaluated in the RI or risk assessment because the facility does not manufacture organic compounds, nor have they been identified in waste streams.

2.2 CRITERIA FOR IDENTIFYING CHEMICALS OF POTENTIAL CONCERN

To meet the objective of only identifying contaminants attributable to the Plant for use in the risk assessment, a COPC was identified if the analyte met all of the following criteria:

- The analyte was detected at least once in a medium at a concentration greater than the background concentration,
- The analyte concentration exceeded one or more RBC and,
- If no RBC exists, and the chemical is known to be an essential nutrient, it was not considered.

The identified COPCs are evaluated in subsequent sections of this risk assessment to determine if they become actual site-specific contaminants of concern (COCs) (as defined in Section 5.0).

2.3 BACKGROUND

Background soil and ground water samples were collected, as well as background gamma radiation measurements, for comparison to Site data.

Background soil and water samples were collected by Monsanto and Kerr-McGee (Dames and Moore 1993). Maximum background values in water were used as screening criteria, and were derived from hydraulically upgradient wells within the flow system of interest (i.e., TW-57, TW-29, and TW-15). Sample sizes for soils ranged from three to twenty samples, using a combined Kerr-McGee/Monsanto data set. Approximately 20 background soil samples were collected from soil types representative of soils around the MCC Plant. The samples were located approximately 5 to 20 miles from the Plant. Details of the background sampling program and the statistical methods used are presented in the Revised Memorandum on Sediment and Soils Investigation (Golder 1993e). Upper tolerance limits (UTLs) based on 95 percent confidence and 95 percent coverage were chosen as the background screening criteria for COPCs, for those chemicals for which 15-20 samples are available. For smaller sample sizes the maximum detected concentration was used (USEPA 1992a).

2.4 DETERMINATION OF RISK-BASED CONCENTRATIONS

RBCs used for offsite media were derived using toxicity values and EPA default exposure assumptions for a residential scenario (USEPA 1991a). The RBCs were calculated based on a target risk of 1-in-10,000,000 (also represented as 1E-07), and a target hazard quotient (HQ) of 0.1 for all media. Screening for COPCs was based on these RBCs. Similarly, RBCs were derived for onsite source materials using industrial scenario default parameters.

RBCs for radionuclides were calculated using the residential and industrial exposure default parameters from Risk Assessment Guidance, Part B (USEPA 1991b), as modified in August, 1992, by EPA Region 10 (USEPA 1992c).

Exposure parameters and toxicity values are presented in Sections 3.0 and 4.0, respectively. Appendix A presents the RBCs used in this risk assessment.

2.5 CHEMICALS OF POTENTIAL CONCERN IN GROUND WATER

Table 2-1 illustrates the screening of ground water for COPCs by comparison with RBCs. These data were derived from the (b) (6) and (b) (6) wells, TW-53, 54, and 55. These offsite wells were evaluated because they are most representative of the future residential scenario (Section 3.0). In addition, chemicals in water were also compared to drinking water standards; i.e., Maximum Contaminant Levels (MCLs), Maximum Contaminant Level Goals (MCLGs), and Secondary Maximum Contaminant Levels (SMCLs).

The COPCs identified in ground water based on the above outlined screening process were cadmium, fluoride, manganese, molybdenum, nitrate as N, selenium, and sulfate. Calcium, iron, magnesium, and potassium were not retained because they are essential minerals and their concentrations are within tolerable levels for humans. Aluminum was dismissed as a COPC because the drinking water SMCL was below background and the maximum detected concentration did not exceed the RBC.

2.6 CHEMICALS OF POTENTIAL CONCERN IN SOILS

Table 2-2 identifies COPCs in offsite soils using the criteria delineated in Section 2.2. All soil data collected under the RI were evaluated. In summary, the COPCs in soil are arsenic, beryllium, cadmium, vanadium, lead-210+D, radium-226+D, thorium-230, and uranium-238+D. Polonium-210 was not evaluated further because it is included as a progeny of lead-210+D (USEPA, 1993). Although iron concentrations exceeded background, it was not retained because it is an essential nutrient and was not present at levels considered significantly above background. Aluminum was not retained because of low frequency of exceedance above background and above the conservative screening criteria. Total chromium was assumed to consist of greater than 99 percent chromium III (Golder 1992); hence a chromium III RBC was used for screening. Selenium characterization in soils was limited because of the small quantity (3 samples) gathered late in the RI process.

2.7 CHEMICALS OF POTENTIAL CONCERN IN SOURCE MATERIALS

Table 2-3 identifies COPCs in source materials. The source material risk-based screening concentrations were based on default exposure assumptions for the industrial scenario, (representing the most conservative parameters for evaluating risk). In summary, the COPCs in source materials included arsenic, beryllium, cadmium, fluoride, vanadium, lead-210+D, radium-226+D, thorium-230, and uranium-238+D. Polonium-210 was not retained as a COPC because it is a progeny of lead-210+D. Chromium concentrations in source materials were estimated to be greater than 99 percent chromium III (Golder 1992). Because lead concentrations exceeded its background value and lead is known to be potentially toxic, it could have been retained as a COPC. However, lead was not retained in this risk assessment because of the relatively low magnitude of exceedance in source materials, and the non-exceedance in soils outside of the facility boundary. Lead typically becomes a COPC at approximately 400 mg/kg in soils under residential scenarios (USEPA 1994b).

Table 2-1
Identification of Chemicals of Potential Concern in Ground Water

Analyte	Maximum Concentration[a]	Maximum Background Concentration[b]	MCL	MCLG	Human Health RBC[c]
	(mg/L)				
Aluminum[e]	0.158	0.152	0.05[d]	0.05[d]	3.6E+00
Arsenic	0.0016	0.002	0.05	0.05[f]	1.1E-03
Beryllium	ND	ND	0.001	0	1.8E-02
Cadmium	0.0042	ND	0.005	0.005	1.8E-03
Calcium	235	178	--	--	--
Chloride	166	22	250[d]	--	3.1E+03
Chromium (total)	NE				
Copper	NE				
Fluoride	5.5	0.25	4	4	2.2E-01
Iron	0.594	0.165	--	--	--
Lead	NE				
Magnesium	133	106	---	--	--
Manganese	0.05	0.010	---	--	1.8E-02
Molybdenum	0.126	ND	---	--	1.8E-02
Nickel	0.01	ND		0.1[f]	7.3E-02
Nitrate as N	12.2	1.44	10	10	5.8E+00
Potassium	16	5.85	--	--	--
Selenium	0.472	0.0075	0.05	0.05	1.8E-02
Silver	NE				
Sulfate Ion	478	110	250[d]	500[f]	--
Vanadium	0.018	0.014	--	--	2.6E-02
Zinc	0.216	0.011	5[f]	--	7.3E-01
RADIONUCLIDES	(pCi/g)				
Radium-226+D	NE				
Radium-228+D	NE				
Radon-222	NE				
Uranium-238+D	NE				

Shading indicates maximum detected concentration exceeds column values; shading in the Analyte column indicates a COPC.

a = Ground water wells evaluated include: (b) (6) TW-53,54,55. These wells would represent ground water potentially available in a future residential scenario.
b = Based on Wells TW-57, TW-29, TW-15 from November 1992 and May 1993 sampling records.
c = RBC based on residential default exposure parameters.
d = Secondary MCL
e = Aluminum is not retained because SMCL is below background, and max is less than RBC
f = Proposed
ND = Not detected
NE = Not evaluated; dropped as a COPC during Phase I or early Phase II risk screening analysis.

Table 2-2
Identification of Chemicals of Potential Concern in Soil

Analyte	Maximum Concentrations	Background		Noncancer RBCs[b] HQ=0.1	Carcinogenic RBCs[b] Risk = 1E-07
		Maximum	UTL[a]		
	(mg/kg)				
Aluminum	30200[h]	16,500	19,187	27,000	--
Arsenic	34.0	5.4	6.1	8.2	3.7E-02
Beryllium	4.0	1.7	2.7		1.5E-02
Cadmium	168	9.7	9.3	27	--
Chromium Total	325	21.0	23.3	27,000[g]	--
Copper	42	19.6	23.1	1,000	--
Fluoride[c]	136.0	6.1	4.9[h]	1,600	--
Iron	55,500	23,000	NC	--	--
Lead	68	81	NC	--	--
Manganese	1380	696	807	3,800	--
Molybdenum	2.9	1.7	1.6	140	
Nickel	87.3	53	NC	550	--
Nitrate as N	47	13	NC	44,000	--
Selenium[d]	109	0.4	NC	140	--
Silver	13.0	1	NC	140	--
Uranium[e]	5.3	0.5	NC	90	
Vanadium	467	42	36.0	190	--
Zinc	2,670	123	112.6	8,200	--
RADIONUCLIDES	(pCi/g)				
Lead-210+D	65	3.2	4.0		1.2E-01
Polonium-210[f]	77	3.8	3.7		5.3E-01
Potassium-40	19	20	NC		7.7E-03
Radium-226+D	17	2.7	2.9		6.9E-03
Radium-228+D	1.4	1.7	NC		1.4E-02
Thorium-228+D	1.6	1.6	1.7		7.4E-04
Thorium-230	18	2.1	2.1		5.7E+00
Thorium-232	1.6	1.7	NC		6.4E+00
Uranium-238+D	16	1.4	2.4		1.1E-01

Shading indicates maximum detected concentration exceeds column values; shading in the Analyte column indicates a COPC.

a = Log normal conversion before UTL calculation.
b = Based on residential default exposure parameters.
c = Site fluoride concentrations based on soluble fraction only.
d = Limited data set.
e = Uranium concentration converted from measured activity of U-238 by multiplying by 0.331.
f = Polonium-210 not retained because it is considered in lead-210+D chain.
g = Chromium III RBC
h = Outlier eliminated from data set before treatment of data set
NC = Not calculated.

Table 2-3
Identification of Chemicals of Potential Concern in Source Materials

Analyte	Maximum Concentrations	Background Concentration		Noncancer RBCs[g] HQ=0.1	Carcinogenic RBCs[g] Risk=1E-07
		Maximum	UTL[a]		
	(mg/kg)				
Aluminum	27,700	16,500	19,187	200,000	--
Arsenic	500	5.4	6.1	61	3.3E-01
Beryllium	60.1	1.7	2.7	1,000	1.3E-01
Cadmium	2,070	9.7	9.3	100	--
Chromium (Total)	30,500	21.0	23.3	200,000[b]	--
Copper	86.9	19.6	23.1	7,600	--
Fluoride[c]	14,500	66.1	11.6	12,000	--
Iron	12,200	23,000	NC	--	--
Lead	200	81	NC	--	--
Manganese	899	696	807	29,000	--
Molybdenum	893	1.7	1.6	1,000	
Nickel	170	53	NC	4,100	--
Nitrate as N	79	13	NC	330,000	--
Selenium[d]	231	0.4	NC	1,000	--
Silver	94	1	NC	1,000	--
Uranium[e]	15.6	0.5	NC	6700	--
Vanadium	65,100	42	36.0	1,400	--
Zinc	54,200	123	112.6	61,000	--
RADIONUCLIDES	(pCi/g)				
Lead-210+D	390	3.2	4.0	--	4.9E-01
Polonium-210[f]	260	3.8	3.7	--	2.1E+00
Potassium-40	11	20	(NC)	--	4.0E-02
Radium-226+D	54	2.7	2.9	--	3.6E-03
Radium-228+D	1.0	1.7	(NC)	--	7.5E-03
Thorium-228+D	0.9	1.6	1.7	--	3.9E-03
Thorium-230	430	2.1	2.1	--	2.3E+01
Thorium-232	4.8	1.7	(NC)	--	2.6E+01
Uranium-238+D	48	1.4	2.4	--	5.7E-01

Shading indicates maximum detected concentration exceeds column values; shading in the Analyte column indicates a COPC.

a = Log normal conversion before UTL calculations.
b = Chromium III RBC
c = Site Fluoride concentrations based on soluble fraction only.
d = Selenium date may be unreliable, awaiting further sample results.
e = Uranium concentration converted from measured activity of U-238 by multiplying by 0.331.
f = Polonium-210 not retained because it is considered in lead-210+D chain.
g = Risk-based concentrations are based on the industrial scenario default exposure factors
NC = UTL not calculated

2.8 CHEMICALS OF POTENTIAL CONCERN IN AIR

The COPC list was derived from the chemicals modelled in the Air Dispersion Modelling report (SENES 1993). The chemicals modelled in the report were presumed to be COPCs because they are COPCs in soils, which have resulted from air deposition. Other gasses and vapors associated with MCC Plant activities were not considered site-related COPCs, and thus, were not modelled. Particulate matter (PM-10) was modelled, but was not considered a COPC, because the facility is in compliance with PM-10 standards and PM-10 represents a physical rather than chemical-specific hazard. Chemicals modelled but not retained for the risk assessment include silver, vanadium, fluoride, molybdenum, and zinc. These chemicals were not evaluated because of lack of toxicity data for the inhalation pathway.

Extrapolation of inhalation reference doses (RfDs) from oral RfDs was not considered necessary for the COPCs at this site. EPA concluded that risks associated with those airborne COPCs for which inhalation toxicity criteria are unavailable would only make a small contribution to the total site risk via the air pathway. The chemicals evaluated (arsenic, beryllium, cadmium, lead-210+D, radium-226+D, thorium-230, and uranium-238+D) were presumed to pose the vast majority of risk in the air pathway because of known carcinogenic properties, and should provide adequate information for decision makers.

Because radium-226 concentrations were elevated in source materials and some soil samples, radon-222 (or decay product [gas] of radium in soils) concentrations may be elevated in air around some areas of the Plant. However, empirical data have not been collected, and concentrations have not been modelled as part of the RI.

2.9 GAMMA RADIATION

Several radionuclides were considered COPCs in soil, source materials, and air. Because these COPCs emit gamma radiation, gamma measurements were performed as part of the remedial investigation. This information was used to provide (with respect to background) a comparison of potentially elevated gamma radiation levels with known elevated levels of radionuclide COPCs. Gamma measurements obtained at all source pile locations exceeded the background gamma UTL (Table 2-4). None of the soil gamma measurements exceeded the background gamma UTL.

Because exposure to gamma radiation may be of concern, industrial exposure and risk related to gamma irradiation was further evaluated in this assessment and presented in Appendix E.

2.10 SUMMARY OF CHEMICALS OF POTENTIAL CONCERN

Table 2-5 illustrates the final list of COPCs in all media. Many of these chemicals were evaluated further depending on the specific exposure scenarios developed in Section 3.0.

Data gaps or uncertainties include the limitations in soil selenium data, as well as the failure to measure or model radon-222 concentrations in air.

Table 2-4
Identification of Gamma Radiation Levels Exceeding Background

Media	Reading (μR/hr)		Background (μR/hr)	
	AVG.	MAX.	UCL[a]	UTL[b]
Offsite soils (n=41)	18.0	23.1	19.8	23.7
Source Piles (N ≈ 9 -30)				
Nodules	94.5	117.1	19.8	23.7
Slag	70.8	111.3	19.8	23.7
Treater Dust	69.7	80.2	19.8	23.7
Baghouse Dust	35.0	38.1	19.8	23.7
Underflow Solids	46.8	60.5	19.8	23.7

Shading indicates exceedance of background.
a = UCL is a 95% upper confidence limit of the mean (n=60)
b = UTL based on 60 background gamma readings; Coverage = 95%, Confidence = 95%

Table 2-5
Summary of Chemicals of Potential Concern (COPCs) in Source Materials, Soils, Ground Water and Air

COPC	Sources	Offsite Soils	Groundwater	Air
Arsenic	X	X		X
Beryllium	X	X		X
Cadmium	X	X	X	X
Fluoride	X		X	
Manganese			X	
Molybdenum			X	
Nitrates			X	
Selenium			X	
Vanadium	X	X		
RADIONUCLIDES				
Lead-210	X	X		X
Radium-226+D	X	X		X
Thorium-230	X	X		X
Uranium-238+D	X	X		X

3.0 EXPOSURE ASSESSMENT

This exposure assessment identifies the sources and mechanisms of chemical releases; evaluates potential pathways and populations exposed to COPCs; and quantifies the magnitude and duration of potential exposure to those chemicals. Exposure is quantified by developing a reasonable maximum exposure (RME) scenario, which is a conservative exposure case still within the range of possible exposures. In addition, an average exposure (AVG) scenario is presented in the residential scenarios to reflect more typical exposures.

Exposure assumptions used in this risk assessment were primarily based on EPA Superfund standard default factors (USEPA 1989a, 1990b, 1991c), except where tailored to meet specific site conditions. Standard default factors describe the intake rate, exposure frequency, and duration of an exposed individual under an RME scenario. These factors describe patterns of exposure that are above average, and are selected to be protective of individuals in the exposed population who have high contact rates and/or long exposure frequency and duration. For example, the RME describes a resident living at the same location for 30 years.

The exposure assessment generally characterizes the intakes of COPCs and external exposure to radionuclides. Intake and exposure factors are developed, which are applied to chemical- and radionuclide-specific parameters to calculate hazards and risks.

Exposure point concentrations are also developed in this section. The method for summarizing data is described, as well as the specific sampling locations used to calculate these exposure concentrations. The following concentration and exposure estimates were used:

Industrial	RME concentrations/activity using default RME exposure assumptions (USEPA 1991c). Site-specific assumptions were used when additional exposure information was available.
Residential	RME or AVG concentrations/activity using default RME or AVG exposure assumptions, respectively.

Although regulatory decisions for Superfund sites are based on RME exposures, average exposure factors are presented for comparative purposes (residential scenarios only) and semi-quantitative evaluation of uncertainties. For this risk assessment, it was assumed that no additional transport, degradation, or attenuation of environmental contaminants would occur over time. Additionally, because reference doses and slope factors (Section 4.0) are based on administered, rather than absorbed doses, no adjustments were made for matrix effects on absorption of COPCs.

3.1 EXPOSURE PATHWAY ANALYSIS

Exposure pathways are defined as those specific mechanisms by which an individual or population is exposed to chemicals present at a site or released from a site. An exposure pathway consists of four elements: 1) a source and mechanism for direct exposure or release

of a chemical into the environment; 2) an environmental transport medium (air, soil, and water); 3) a point or site of potential human contact with a medium (exposure point); and 4) a human exposure route (ingestion, inhalation, dermal contact). The total exposure of an individual or population may be a composite of several separate exposure pathways.

This section examines the media and exposure pathways of COPCs. Development of exposure parameters and scenarios is dependent on an evaluation of sources and environmental releases at the MCC Plant.

3.1.1 Ground Water

Contamination of ground water at Monsanto has resulted in the migration of one plume south of the Plant. Monitoring of downgradient water quality is anticipated to continue throughout the RI/FS. Elevated concentrations of some heavy metals and anions are found in the downgradient wells. Currently, there is no human exposure to this water because municipal water is supplied to residents. However, the downgradient (b) (6) well may be used for domestic stock watering or agricultural purposes. A future residential scenario was evaluated in this assessment to evaluate potential health effects due to exposure to contaminants if this ground water was used as a drinking water source.

Contaminated ground water also exists in a secondary plume beneath the hydroclarifier in the central portion of the Plant. Currently, this plume does not migrate beyond the Plant boundary due to extraction of water by the production wells. In the future, the production wells may cease operations, and the contaminants may migrate offsite. Hypothetical future residents could be exposed if ground water from either plume is used for drinking. However, because municipal water is readily available in the area, it is unlikely that future consumption of shallow ground water would occur.

A contaminant plume from the Kerr-McGee Superfund site traverses the southeast perimeter of the MCC Plant. This plume flows primarily in a southwesterly direction. However, based on current hydrogeological data, substantial contaminant mixing from the two sites is not expected (Dames and Moore 1993, Golder 1993c).

In addition, current RI data (Golder 1992; 1993b,c,d) and data from the draft remedial investigation for the Kerr-McGee Superfund site (Dames & Moore 1993) indicate that Formation Springs and Ledger Springs are not impacted by releases from these two facilities.

3.1.2 Surface Water and Sediment

As reported in the Monsanto Site Characterization Summary Report (Golder 1992), storm water runoff does not result in surface water leaving the Plant. It is possible that COPCs in offsite soils are mobilized by snow melt or heavy rains and may enter the Soda Creek watershed.

Monsanto discharges non-contact cooling water from plant operations into Soda Creek under an NPDES permit. Soda Creek water and sediments contain elevated concentrations of chemicals relative to background. These concentrations are at levels which are not thought to be toxic to humans, but may be toxic to other organisms (see Ecological Risk Assessment). In addition,

because the creek is generally not used for recreation, ingestion of Soda Creek water was not considered a pathway of concern. Similarly, human exposure to sediments in Soda Creek is highly unlikely and was not evaluated.

3.1.3 Atmospheric Releases

Particulate emissions from the stacks and source piles result in elevated concentrations of COPCs in air in the vicinity of the Plant, exposing individuals through the inhalation pathway (SENES 1993). In addition, process operations release fugitive dust, gases, and vapors, which contribute to inhalation exposures. Therefore, the air pathway represents a potentially major exposure route and was evaluated in present and future scenarios.

Fugitive dust is released from onsite source piles via wind-blown transport, and is deposited onto soils in adjacent agricultural fields, resulting in potential food chain transfer to humans consuming agricultural products. Information provided in the RI indicates that the dilution of grain with other locally grown grain would mask any potentially detectable increase in contaminant load in grain stocks. The grain pathway is therefore considered a minor exposure route, and was not evaluated in this document.

Windblown contaminants may deposit upon surface water or plant material, resulting in exposure to domestic grazers or local wildlife. Movement of COPCs through the food-chain from wildlife (fish or game) to humans is possible. However, since deer and other game animals infrequently visit the Plant vicinity and no viable fisheries exist near the Plant, this pathway was also considered minor. In the recent past, cattle were pastured in fields south of the facility; currently, grazing is limited to a few horses. Although cattle grazing could potentially occur in the future, it is unlikely that a single individual would consume beef or dairy products from these cattle as their single source of these products. Thus, any biotransfer through beef or dairy to humans is considered a minor exposure route, and was not considered in this risk assessment.

3.1.4 Soil and Source Material

Releases from MCC operations have resulted in elevated concentrations of COPCs in soils. Individuals currently exposed to soil would include residents near the Plant. Such human exposure includes inadvertent ingestion of soil, inhalation of soil particulates, indirect exposure by ingestion of garden produce grown in affected soil, and external exposure to radionuclides. Future residential use of land north and south of the Plant could occur, with similar exposure pathways. Exposure to releases from source materials and waste piles were evaluated. The pathways of exposure for these waste piles are the same as those for soil: ingestion, inhalation, and external exposure.

A major radioactive decay product of radium-226 is radon-222+D. The natural release of radon-222+D from radium-226 in soil or source materials may cause an increase in atmospheric concentrations and hence exposure via the inhalation pathway. Various modelling approaches predict radon concentrations in air, as a function of radium-226+D concentrations in soils or other solid media. Such modelling techniques range in complexity from a volatilization factor approach presented in EPA (1991b), to sophisticated modelling approaches such as RESRAD (USDOE, 1993).

For the Monsanto Plant, the volatilization factor approach would excessively overestimate atmospheric radon concentrations (due to local atmospheric conditions, depth of contamination in the media, and the texture and porosity of source materials). RESRAD would more accurately estimate radon exposure; however, such complex radon modelling is out of the scope of this assessment. In addition, because no empirical radon data exists, a data gap is apparent. However, since potential exposure to radon is often linked to radium-226+D in soils or source materials, radon exposures will be qualitatively inferred from radium exposure. Additional discussion regarding the limitations of radon quantification is provided in Section 5.6.3.

3.1.5 Gamma Radiation

Gamma radiation has been measured at various source pile areas at levels exceeding background. Because gamma radiation in sufficient quantities may affect human health and many sampling locations exceed background levels, gamma radiation emanating from the radionuclide COPCs was also considered to be of concern. Exposure was further analyzed in Appendix E for current and future industrial scenarios.

3.2 CONCEPTUAL SITE MODEL

Based on the information presented above, a model was developed to identify the media and exposures of concern at the MCC Plant.

The conceptual site model presented in Figure 3-1 summarizes contaminant sources, environmental pathways to exposed individuals, and routes of entry into the body for each medium of exposure. The primary pathways are those exposure routes that are assumed to be of greatest concern, contrasted with the minor pathways which were not evaluated. Although minor pathways do exist, evaluation of the major pathways should provide risk managers with sufficient information with which to make remedial decisions.

The media of concern within the Plant boundary are source materials, road dusts, and air. Outside the Plant boundary the media of concern are soils, ground water, and air. Ground water samples were used to estimate future exposures to contaminated water. The concentrations of chemicals in air were based on an air modelling assessment (SENES 1993). Table 3-1 summarizes the exposure media for each scenario. The following sections briefly explain all scenarios evaluated in this risk assessment.

Table 3-1
Exposure Media

Scenario	Ground Water	Air	Soil	Source Material
Industrial - Current and Future	No	Yes	No	Yes
Current Residential	No	Yes	Yes	No
Future Residential	Yes	Yes	Yes	No

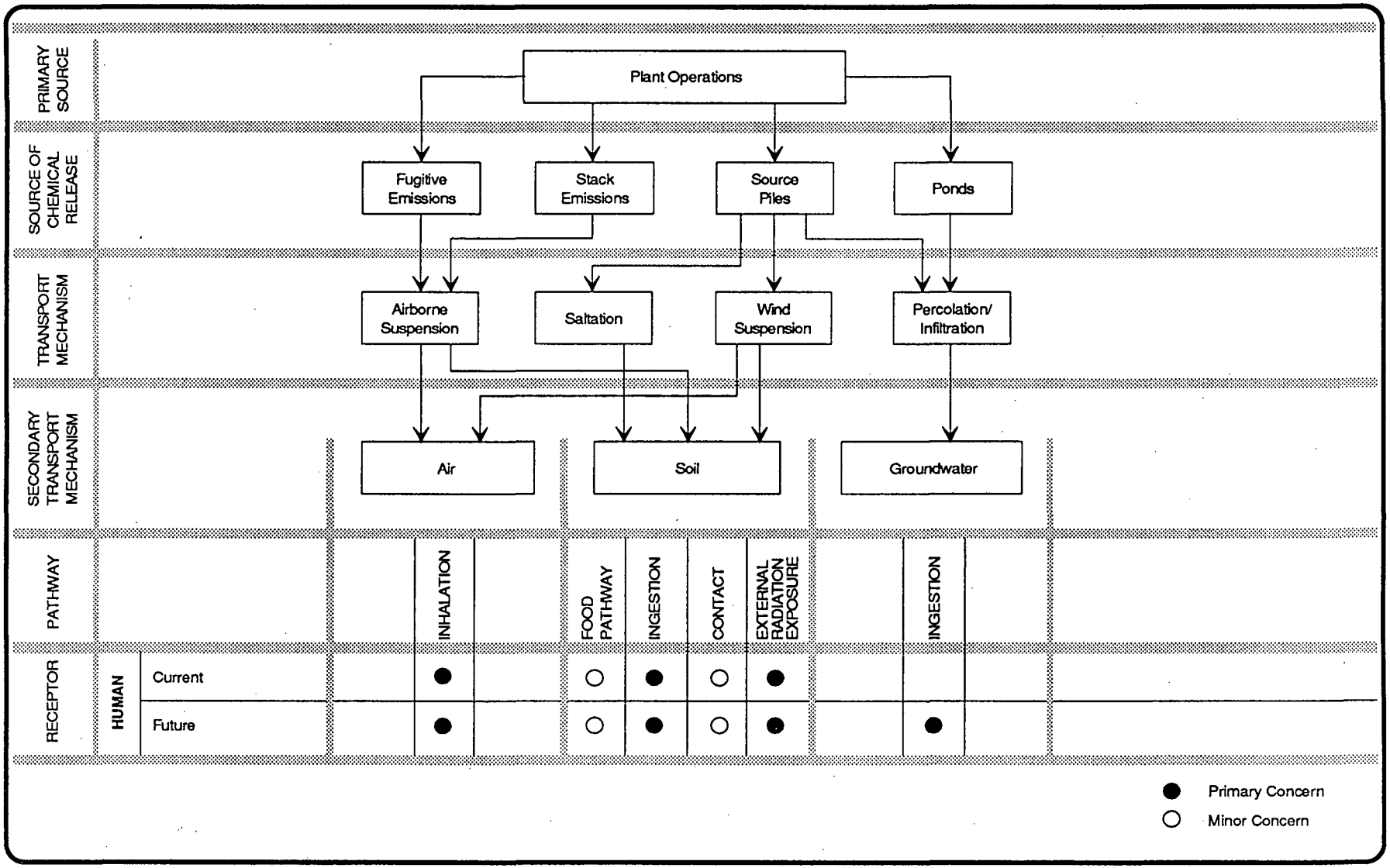

Figure 3-1

CONCEPTUAL SITE MODEL
MONSANTO FACILITY
SODA SPRINGS, IDAHO

3.3 DEVELOPMENT OF EXPOSURE SCENARIOS

The development of exposure scenarios was based on the conceptual site model, site visits to the Monsanto Plant, information submitted through the RI, and Superfund risk assessment guidance. Superfund guidance recommends evaluation of current and future scenarios to evaluate potential exposures to COPCs in the absence of remediation (USEPA 1991c). Several scenarios were analyzed to represent exposures: current and future industrial within the active facility, current residential to the northwest, west, and south, and future residential to the north and south. Evaluation of worker exposure to contaminants inside the various buildings and structures throughout the facility is not within the scope of this baseline risk assessment.

Other potential exposure scenarios, such as recreational and agricultural activities, were considered to be of only minor importance for this site. This is because of the local conditions such as length of growing season, weather, and altitude, and because the residential scenario used is considered sufficiently conservative without consideration of these additional pathways.

3.3.1 Current Industrial Scenario

Populations exposed to COPCs in source piles and airborne dusts include individuals working at or visiting the facility. MCC has provided worker time and motion data (Monsanto 1993) which was used to describe current exposures.

An effort has been made to quantify worker exposures to releases of hazardous substances from source piles and waste materials. The presence of multiple source areas and the nature of worker exposures has complicated the development of a reasonable subset of exposure scenarios. There are several tasks that workers perform in the course of their careers, which may cause them to be exposed to different source areas for varying durations. For example, an individual who drives the water truck (who is potentially exposed to contaminants associated with road dusts) may only perform this task for a portion of their entire career at Monsanto. Due to the seniority system, the individual may transfer to another job within the plant and be potentially exposed to contaminants associated with a different on-site location (e.g., at the slag pile). As a simplifying assumption, it was assumed that a worker is exposed to a single waste or source material for the entire duration of their career at Monsanto (assumed to be 25 years).

For this assessment, exposure at each source of contamination was evaluated separately. Reasonable maximum exposures were evaluated by choosing source areas where relatively high concentrations occur. The areas also were selected based on the conceptual site model, site-specific exposure information provided by Monsanto, and COPC concentrations in the source materials. Specific sources evaluated included the underflow solids, nodules, treater dusts, slag, baghouse dusts, as well as road dusts. The current industrial scenario evaluated exposure based on modified default exposure assumptions (Table 3-2a).

The number of hours per day was used to modify the source material ingestion rate, inhalation rate, and the gamma exposure factor. The extent to which workers are shielded from external gamma radiation was determined by evaluating the time and motion information and empirical gamma shielding data provided by Monsanto. For example, the potentially maximally exposedworkers at the baghouse dusts and nodules areas were assumed to be outdoors for 1.5

Table 3-2a
Exposure Assumptions for the Current Industrial Scenario

Source Material	Task	Hours/day	Days/year	Shielding Factor (unitless)
Baghouse Dusts	Reclaim operator	1.5	250	0
Nodules	Bin operator	1.5	250	0
Slag	Pot carrier operator	4	250	0.45
Road Dusts	Water truck operator	6	160	0.45
Treater Dusts	Loader operator	1	250	0.45
Underflow Solids	Loader operator	6	180	0.45

hours per day (Montgomery-Watson 1994) and unshielded from gamma radiation. However, workers at all other outdoor locations were assumed to be located inside heavy vehicles and partially shielded from radiation. The derivation of gamma shielding factors is presented in Table 3-2b). A shielding factor (Se) of 0.45, the 95th percentile value, was assumed for all workers within heavy vehicles. Other less significant exposure scenarios (e.g., office worker, or a worker exposed to a less contaminated source area) were not developed, because it was presumed that the evaluation of reasonable maximum exposures to the major source areas would provide sufficient information for remedial decision makers.

3.3.2 Future Industrial Scenarios

A future industrial scenario was developed to estimate potential future risks within the Plant boundary. This scenario assumes that the Monsanto Plant would not be operating. However, other industries or businesses may operate; consequently, the potential for exposure to source materials does exist in the future. However, such a scenario is unlikely in the near future because Monsanto has estimated the facility will continue to operate for a few more decades.

Exposure to contaminants in ground water beneath the plant was not evaluated. Use of this water as drinking water is unlikely because the plant is expected to continue to operate on site, and municipal and upgradient water sources are available for plant use.

The airborne COPCs, as described in the air model with and without stack emissions, were used to evaluate the significance of the future air pathway. Exposure parameters used for the future industrial scenario are provided in Table 3-3.)

3.3.3 Current Residential Scenario

The current residential scenario was used to estimate potential exposure to residents who currently reside near the Plant, or within areas potentially impacted from site operations. The closest current resident lives approximately one-half mile west of the Plant. Exposure parameters used to evaluate this scenario were based on standard EPA default RME and average exposures for soil ingestion, inhalation of dusts, and external gamma exposure.

Drinking water supplies for current residences are not affected by the site. Therefore, this scenario does not include exposure to contaminants in ground water.

A garden produce ingestion scenario was also considered for the conceptual model of exposure to residents. Many residents in the Soda Springs area currently cultivate gardens during at least some portion of the year. Biotransfer of COPCs through home-grown produce may represent an exposure pathway under certain scenarios. The significance of this pathway is highly dependent on a number of factors. Chemical species/speciation, plant species and parts consumed, soil type, cultivation method, length of growing season, availability of commercially grown produce, and local diet are all factors which could substantially influence exposure. Quantification of exposure would require assumptions regarding all of these highly variable factors. The high degree of uncertainty involved in making these assumptions for this risk assessment precludes credible quantification; therefore, this pathway was considered qualitatively.

Table 3-4 summarizes exposure factors used to calculate risk for the residential scenarios.

Table 3-2b
Shielding Factors for Heavy Vehicle Operators

Dose Rates (μrem/hr)[a]		Dose Reduction Factor[b]
Unshielded	Shielded	
48	22	0.38
45	15	0.23
35	11	0.17
45	17	0.28
45	25	0.49
53	21	0.32
60	40	0.63
65	15	0.15
40	15	0.26
43	22	0.43
52	15	0.20
60	17	0.20

95th percentile Dose Reduction Factor (DRF) = 0.55
95th percentile Shielding Factor (i.e., 1-DRF) = 0.45

(a) Shielding data presented in Montgomery-Watson (1994).

(b) See text for DRF calculation.

Table 3-3
Future Industrial Scenario Exposure Factors

	RME Exposure Factors	
Exposure Route	Noncarcinogens	Carcinogens
Soil Ingestion		
Ingestion Rate (mg/day)	50	50
Exposure Frequency (days/year)	150	150
Exposure Duration (years)	25	25
Body Weight (kg)	70	70
Averaging Time (days)[a]	9,125	25,550
Dust Inhalation		
Inhalation Rate (m^3/day)	20	20
Exposure Frequency (days/year)	250	250
Exposure Duration (years)	25	25
Body Weight (kg)	70	70
Averaging Time (days)[a]	9,125	25,550
External Exposure to Radionuclides		
Gamma Shielding Factor (unitless)	NA	0.0(b)
Gamma Exposure Factor (unitless)	NA	0.24(c)
Exposure Duration (years)	NA	25

(a) Averaging time for noncarcinogens is the exposure duration x 365 days/yr. For carcinogens it is 70 years x 365 days/year.

(b) Workers assumed to be unshielded in the future industrial scenario.

(c) Default value

NA = not applicable

Table 3-4
Default Residential Scenario Exposure Factors

Route	RME Exposure Factors		AVG Exposure Factors	
	Noncarcinogens	Carcinogens	Noncarcinogens	Carcinogens
Soil Ingestion				
Intake Factor	114 (mg-yr/kg-day) (integrated)[a]	114 (mg-yr/kg-day) (integrated)	100 mg/day	100 mg/day
Exposure Frequency	350 days/year	350 days/year	275 days/year	275 days/year
Exposure Duration	I	I	9 years	9 years
Body Weight	I	I	70 kg	70 kg
Averaging Time[b]	10,950 days	25,550 days	3,285 days	25,550 days
Dust Inhalation				
Intake Rate	20 m^3/day	20 m^3/day	20 m^3/day	20 m^3/day
Exposure Frequency	350 days/year	350 days/year	275 days/year	275 days/year
Exposure Duration	30 year	30 year	9 year	9 year
Body Weight	70 kg	70 kg	70 kg	70 kg
Averaging Time	10,950 days	25,550 days	3,285 days	25,550 days
Water Ingestion				
Intake Rate	2 l/day	2 l/day	1.4 l/day	1.4 l/day
Exposure Frequency	350 days/year	350 days/year	275 days/year	275 days/year
Exposure Duration	30 year	30 year	9 year	9 year
Body Weight	70 kg	70 kg	70 kg	70 kg
Averaging Time	10,950 days	25,550 days	3,285 days	25,550 days
External Exposure To Radionuclides				
Gamma Shielding Factor (Se)	NA	0.2	NA	0.2
Gamma Exposure Factor (Te)	NA	1.0	NA	1.0
Exposure Duration	NA	30 Year	NA	9 Year

a = Based on intake rates of 200 mg/day child and 100 mg/day adult and body weight child of 15 kg and body weight adult of 70 kg.

b = Averaging time for noncarcinogens is the exposure duration times 365 days/year. For carcinogens it is 70 years times 365 days/year.

I = Integrated into Intake Factor (USEPA 1989a).

3.3.4 Future Residential Scenarios

The future residential scenario was used to estimate risks posed to potentially exposed populations in the future. In this scenario, the following exposure pathways were considered using standard EPA default exposure parameters: soil ingestion, ingestion of ground water, inhalation of particulates in air, and external gamma exposure. As with the current residential scenario, the garden produce scenario was considered qualitatively. An integrated approach for soil ingestion was used, assuming one ingestion rate throughout childhood and another throughout adulthood (USEPA 1991b).

Future residential scenarios were evaluated for four locations: one at the southern fenceline, another further south of the Plant and two north of the Plant. One hypothetical future resident was assumed to reside south of the MCC property and within the residential zoning area of Soda Springs. One future resident was assumed to be located northwest of the Plant along a transportation corridor. For conservatism, another future resident was assumed to be located off the northern fence line of the Plant. The northern residents were not evaluated for the ground water ingestion pathway, because the residences are upgradient of the contaminant plume. The future Southern I residents were evaluated for ground water consumption under two scenarios; one scenario assumed production well shut-down, and the other scenario assumed continued production. This evaluation was considered protective of the southern fenceline resident. Exposure parameters for the future residential scenario are listed in Table 3-4.

It is considered very unlikely that the land within the operating facility boundary would ever be used for residences. Therefore, no exposure calculations were developed for a residential scenario within the Plant boundary. Residential risks would be considerably higher than industrial risks because of greatly increased potential exposure.

3.4 EXPOSURE POINT CONCENTRATIONS

Exposure point concentrations (EPCs) for COPCs at the Monsanto Plant were derived in a manner consistent with EPA guidance to evaluate reasonable maximum exposures. For chronic toxicity hazards and carcinogenic risks, RMEs are based on exposures to upper-bound estimates of mean concentrations over the exposure period. The lesser value of either the 95% upper confidence limit (UCL) on the mean or the maximum concentration was used as the EPC. For this site, the sample size was generally small ($n<5$), and the maximum was below the UCL in all cases, so the maximum value was used as the EPC. Average (AVG) estimates (used in residential scenarios only) were based on the arithmetic mean concentration of chemicals in each medium. Chemical data for various media are presented in the Preliminary Site Characterization Summary Report (Golder 1992) and in various remedial investigation technical memoranda (Golder 1993a,b,c,d,e).

EPCs at background were also determined for the purposes of calculating hazards and risks at background. These values were determined in the same manner as Site-related EPCs, except that a log-normal distribution was assumed. Because of the larger background sample size, UCLs were selected to represent the EPCs. It should be noted that background EPCs are different from background screening criteria. Background EPCs represent an upper-bound estimate of the

average background concentrations; whereas, the background screening values (UTLs) represent an upper-bound estimate of individual sample concentrations. UTL screening criteria are expected to be higher than background EPCs.

Air concentrations for all exposure scenarios were derived from the Air Dispersion Modelling Report (SENES 1993). The 1991 air dispersion data presented in this report were used. However, for the current scenarios, the data were modified to reflect the effect of frozen ground and snow cover on wind-blown emissions and the fraction of total suspended particulates (TSP) comprised of respirable PM-10 (SENES 1994a,b). For future scenarios, the data were modified to account for the lack of process-related activities in the hypothetical circumstance of plant shut-down. Consequently, future air concentrations reflect emissions solely from fugitive dust generation (corrected for snow cover/frozen ground and PM-10).

3.4.1 Industrial Scenarios

The EPCs for the industrial scenarios (current and future) are represented by the maximum concentration (or UCL) of each COPC for each applicable source material which characterizes the area of exposure (e.g., the slag pile area, the underflow solids area, or road dusts). Figure 3-2 shows the sampling points used to calculate the EPCs for the current and future industrial scenarios. Road dust sampling locations are not shown on Figure 3-2; however, road dusts were collected on the east service,west service, ore, quartzite, and slag roads. EPCs for the industrial scenarios are listed in Table 3-5.

3.4.2 Residential Scenarios

Figures 3-3 and 3-4 show sampling locations for data used in current and future residential scenarios, respectively. For current scenarios, the nearest three soil locations and modelled discrete air receptor locations were used to calculate EPCs at three residences. Subsequent to the start of the remedial investigation, Monsanto acquired the property (and former residence) at the southern fenceline. The future residential scenario used the southern fenceline location (South I) in addition to three locations around the facility to show a range of potential future risks beyond the Plant boundary. In addition to soil and air data, ground water was evaluated for the future southern scenario at South II. Offsite well data from TW-53,54,55 were used assuming continued pumping from the production well. Onsite well data from TW-40,42,43 were used assuming production well shutdown.

As with the industrial scenario, RME concentrations were represented by the maximum concentration. Average (mean) concentrations were also calculated for an average exposure scenario. EPCs are presented in Table 3-6.

3.4.3 Uncertainties Related to Exposure Point Concentrations

The number of samples used to develop the EPCs in the various media for each scenario was generally small (usually 1 to 6 samples). Small sample sizes introduce increased uncertainty when determining the variability of concentrations in the environmental media. Exposure to soil in the residential scenarios is based on the nearest three soil samples, which may be several hundred feet apart from each other and the resident. Because the RME concentration generally relied on the maximum of the three samples, EPCs may either over- or under-estimate actual exposures.

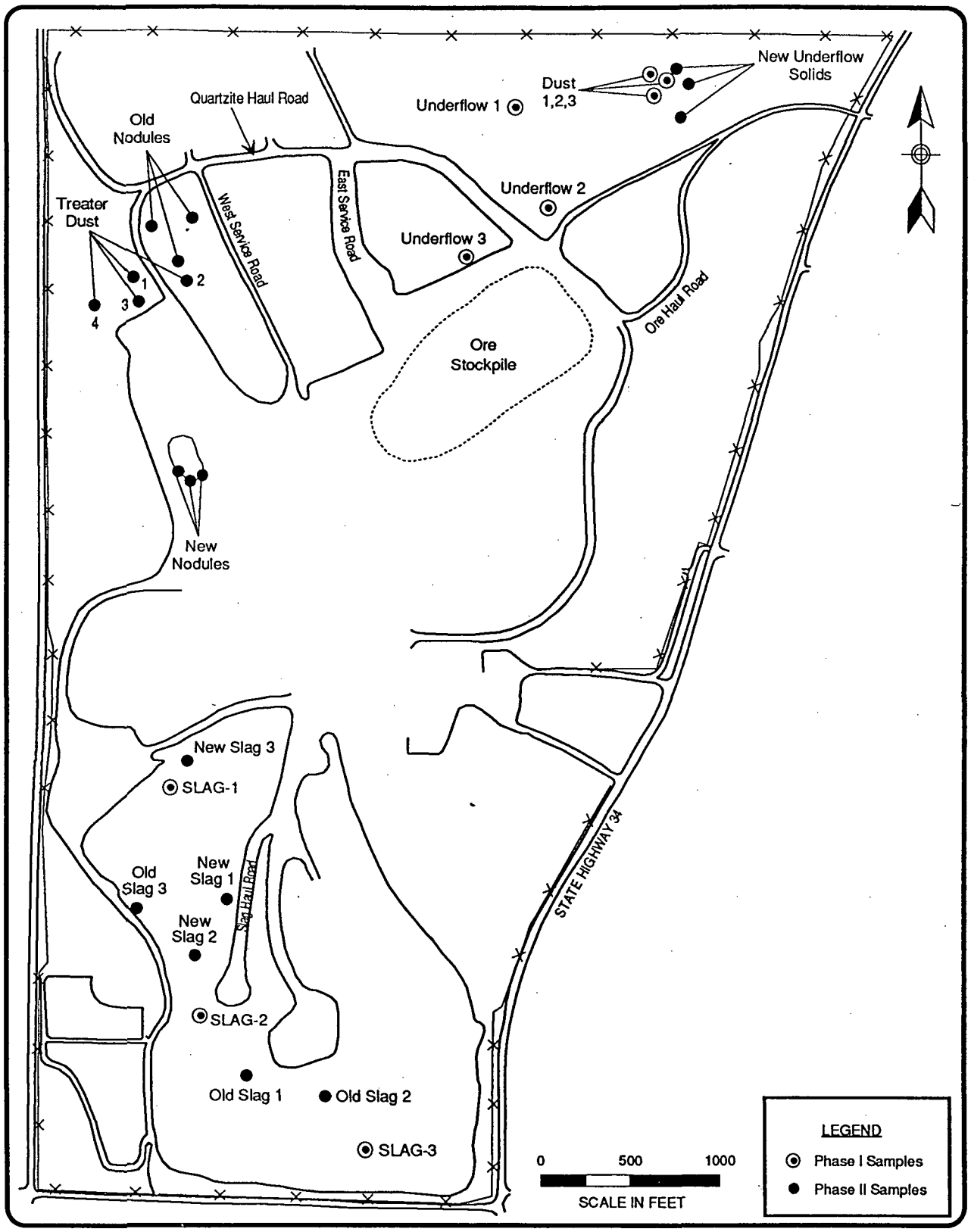

Figure 3-2

SELECTION OF SOURCEPILE SAMPLES USED IN CALCULATING ON-SITE INDUSTRIAL EXPOSURES

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Table 3-5
Exposure Point Concentrations
Industrial Scenarios

COC	Source Materials (mg/kg or pCi/g)							Air (mg/m^3 or pCi/m^3) All Locations	
	Baghouse Dusts	Nodules	Slag	Road Dusts	Treater Dusts	UFS	Background (soil UCL)	Current	Future
Arsenic	85.0	11.4	6.4	43.0	BB	245.0	4.4	9.7E-07	1.7E-07
Beryllium	4.7	13.7	9.9	3.6	5.9	15.4	1.2	1.5E-07	8.5E-08
Cadmium	504.0	BB	34.5	483.0	134.0	1881.1	2.1	9.9E-06	6.5E-08
Vanadium	769.0	1640.0	524.1	1010.0	844.6	2608.6	25.8	3.7E-05	2.2E-05
Lead-210+D	100.0	BB	6.6	190.0	356.5	340.5	2.5	2.3E-03	1.7E-03
Radium-226+D	32.0	51.2	49.9	42.0	27.0	40.9	1.9	1.0E-03	3.2E-04
Thorium-230	33.0	54.4	50.0	44.0	27.0	44.9	1.4	1.1E-03	3.4E-04
Uranium-238+D	35.0	48.0	44.6	43.0	24.0	43.2	1.7	1.0E-03	3.4E-04

BB = Below background.

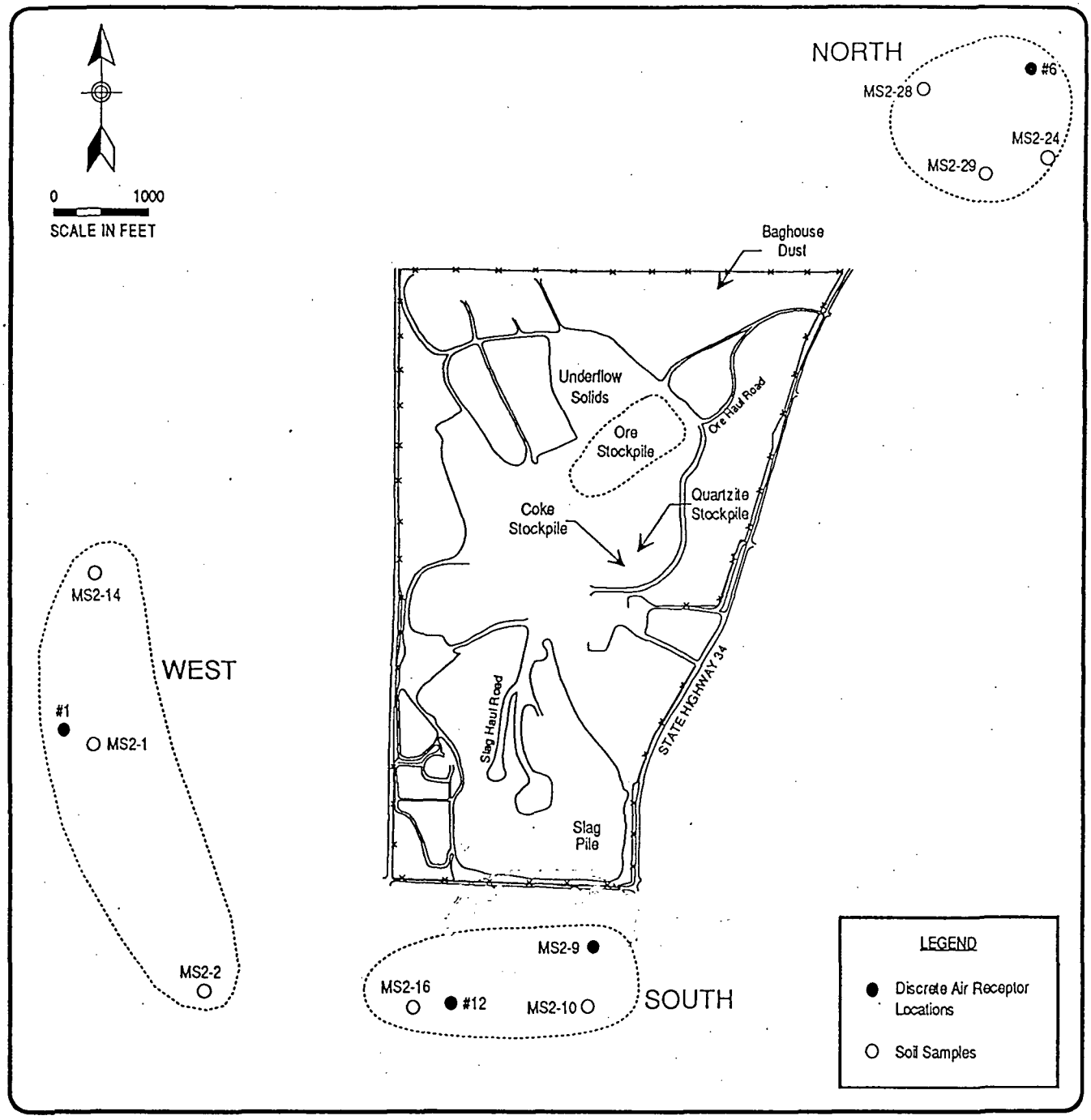

Figure 3-3

ENVIRONMENTAL SAMPLES USED IN CURRENT RESIDENTIAL SCENARIOS

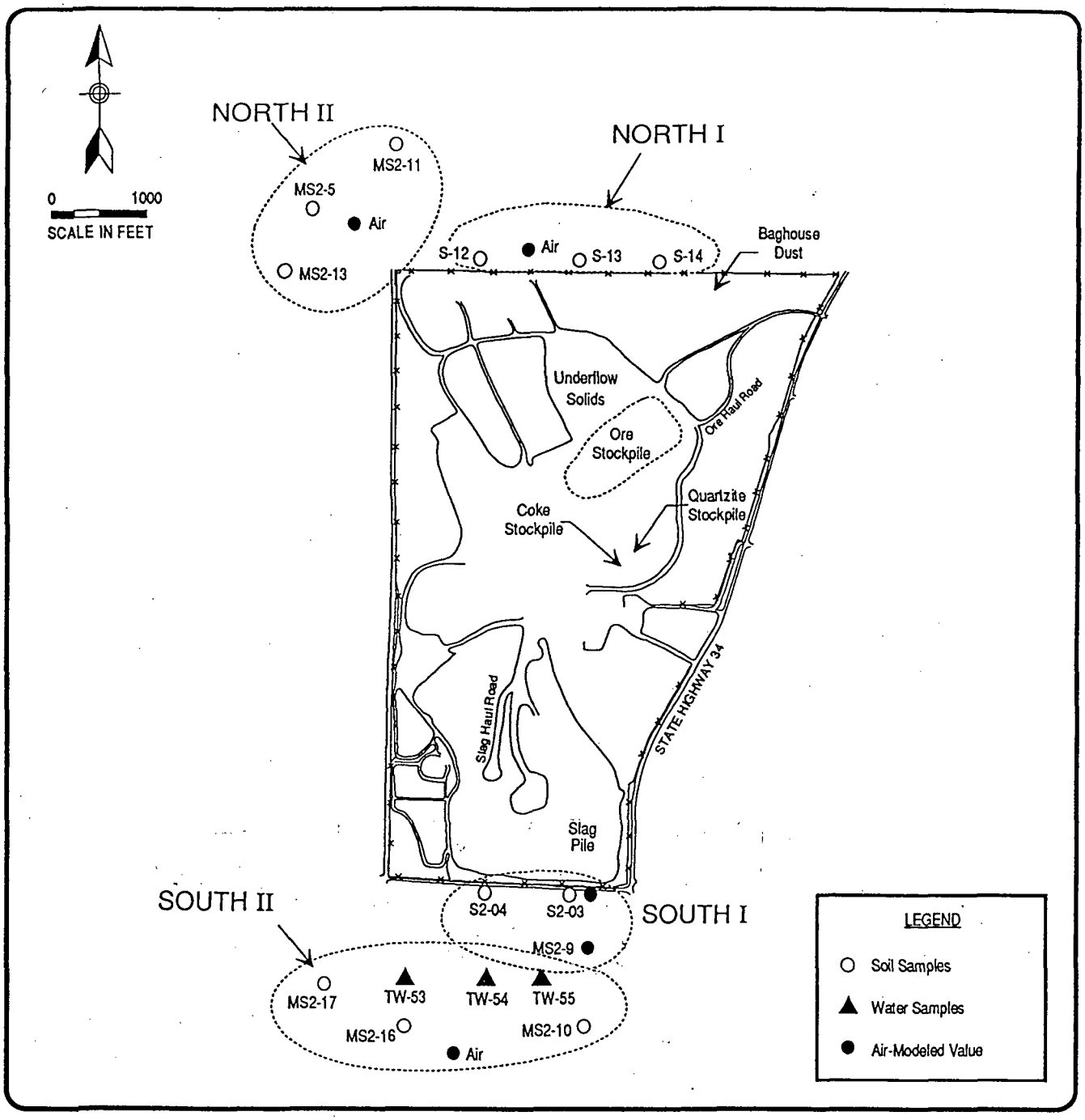

Figure 3-4

ENVIRONMENTAL SAMPLES USED IN FUTURE RESIDENTIAL SCENARIOS

Table 3-6
Exposure Point Concentrations
Residential Scenarios

	Soils (mg/kg or pCi/g)							
	Current (RME)			(Future RME)				Background
COC	South	West	North	South II	North II	North I	South I[a]	(soil UCL)
Arsenic	9	BB	BB	4.9	10.4	34	10	4.4
Beryllium	BB	1.8	1.6	1.4	1.4	3.7	4	1.2
Cadmium	6.4	10.5	BB	6.4	16.1	153	67.6	2.1
Vanadium	40.4	43	BB	37.5	68.3	371	324	25.8
Lead-210+D	2.9	6.6	3.7	2.9	6.9	65	24	2.5
Radium-226+D	BB	BB	BB	BB	2.5	13	12	1.9
Thorium-230	2.1	1.8	1.6	2.1	3.1	12	16	1.4
Uranium-238+D	2.1	BB	BB	BB	2.8	11	10	1.7

	Air (mg/m^3 or pCi/m^3)						
	Current (RME)			Future (RME)			
COC	South	West	North	South II	North II	North I	South I[a]
Arsenic	2.5E-07	7.4E-08	9.1E-08	8.6E-08	4.0E-08	9.3E-07	7.8E-08
Beryllium	6.8E-08	1.6E-08	1.2E-08	1.9E-08	6.7E-09	1.5E-07	1.8E-08
Cadmium	3.3E-06	9.8E-07	1.4E-06	1.1E-06	5.1E-07	1.2E-05	1.0E-06
Vanadium	1.4E-05	3.0E-06	1.6E-06	2.1E-06	1.0E-06	2.0E-05	1.9E-06
Lead-210+D	6.6E-04	2.0E-04	1.6E-04	3.0E-04	1.5E-04	2.9E-03	2.6E-04
Radium-226+D	6.2E-04	1.1E-04	5.7E-05	6.8E-05	2.3E-05	3.9E-04	6.3E-05
Thorium-230	6.5E-04	1.1E-04	6.0E-05	6.9E-05	2.5E-05	4.4E-04	6.5E-05
Uranium-238+D	5.7E-04	1.0E-04	5.4E-05	6.7E-05	2.4E-05	4.4E-04	6.2E-05

	Ground Water (mg/l) Future Southern Residence II (RME)	
COC	Production Well Continues Operation	Production Well Ceases Operation[b]
Cadmium	0.005	2.56
Fluoride	5.1	5.0
Manganese	0.019	0.284
Molybdenum	0.123	0.218
Nitrate	6.42	10.7
Selenium	0.248	0.361

Key:

BB = Below background.

(a) Currently unoccupied home.

(b) Data taken from onsite wells near the hydroclarifier.

Similarly, future consumption of ground water is based on the maximum concentration of three sampling rounds. Based on ground water modelling conducted by Golder Associates (1993c), the uncertainties associated with predicted future concentrations of several COPCs are substantial. In addition, data related to the contaminant plume currently captured by the production wells may not be representative of future downgradient concentrations.

Modelled air concentrations at discrete receptor points representing an average yearly concentration have a large degree of uncertainty. The numerous input parameters used to calculate emissions from source areas and dispersion of particulates each have their own unique uncertainties or error margins. Only one modelled air data point was used in each scenario.

3.5 QUANTIFICATION OF EXPOSURES

Exposure is defined as the contact of an organism with a chemical or physical agent at the exchange boundaries, including skin, lungs or gastrointestinal tract. Estimates of chemical intake (ingestion and inhalation exposure) are derived in accordance with methods developed by EPA.

Exposure is normalized for duration and body weight, and is expressed in units of milligram of chemical administered per kilogram body weight per day of exposure (mg/kg-day). The equations used in this risk assessment for calculating intake factors are presented below.

Soil and Source Material Ingestion. Soil ingestion exposures were evaluated for both adults and children in the future offsite residential scenarios using an integrated approach (USEPA 1989a). Source material and road dust ingestion exposures for onsite workers were also evaluated.

Intake factor estimates for soil ingestion for the residential scenario were determined as follows:

$$\textit{Intake Factor}\ (1/d) = IF \times CF \times EF \times 1/AT$$

Where:

IF = Ingestion Factor (mg-year/kg-day)
CF = Units Conversion Factor (1 kg/1,000,000 mg)
EF = Exposure Frequency (days/year)
AT = Averaging Time (days)

for non-cancer effects: AT = ED in years x 365 days/year
for cancer risk estimates: AT = 70 years x 365 days/year

Where: ED = Exposure Duration (years)

Intake factor estimates for source material ingestion for workers were determined as follows:

$$Intake\ Factor\ (1/d) = IR \times CF \times EF \times ED \times 1/BW \times 1/AT$$

Where:

IR = Intake Rate (mg/day)
CF = Units Conversion Factor (1 kg/1,000,000 mg)
EF = Exposure Frequency (days/year)
ED = Exposure Duration (years)
BW = Average Body Weight (kg)
AT = Averaging Time (days)

for non-cancer effects: AT = ED in years x 365 days/year
for cancer risk estimates: AT = 70 years x 365 days/year

Ground Water Ingestion. Ground water exposure was evaluated for the future offsite residential scenario. Intake factor estimates for water ingestion were estimated with the following equation:

$$Intake\ Factor\ (1/d) = IR \times EF \times ED \times 1/BW \times 1/AT$$

Where:

IR = Intake Rate (L/day)
EF = Exposure Frequency (days/year)
ED = Exposure Duration (years)
BW = Average Body Weight (kg)
AT = Averaging Time (days)

for non-cancer effects: AT = ED in years x 365 days/year
for cancer risk estimates: AT = 70 years x 365 days/year

Inhalation of Dust (Particulates). Inhalation exposures intake factors were estimated for the residential and industrial scenarios with the following equation:

$$Intake\ Factor\ (m^3/kg\text{-}d) - metals = IR \times EF \times ED \times 1/BW \times 1/AT$$
$$Intake\ Factor\ (m^3) - radionuclides = EF \times ED \times IR$$

Where:

IR = Inhalation Rate (m^3/day)
EF = Exposure Frequency (days/year)
ED = Exposure Duration (years)
BW = Average Body Weight (kg)
AT = Averaging Time (days)

for non-cancer effects: AT = ED in years x 365 days/year
for cancer risk estimates: AT = 70 years x 365 days/year

External Exposure to Radionuclides. The external exposure factor for radionuclides was evaluated for all scenarios using the following equation:

$$External\ Exposure\ Factor\ (y) = ED \times (1-Se) \times Te$$

Where:

ED = Exposure Duration (years)
Se = Gamma Shielding Factor (unitless)
Te = Gamma Exposure Factor (unitless)

The gamma exposure factor, or the Te, reflects actual hours and days spent onsite by individuals. The derivation of Te is as follows:

$$Te = (No.\ hours\ per\ day\ onsite/24\ hours) \times (No.\ days\ per\ year\ onsite/365\ days)$$

Ingestion of Radionuclides. The intake factor for ingestion of radionuclides through inadvertent soil ingestion was evaluated for all scenarios using the following equation:

$$Intake\ Factor\ (g) = ED \times CF \times EF \times IR\ (Average\ exposure\ /\ Industrial\ RME)$$
$$Intake\ Factor\ (g) = IF \times EF \times CF\ (RME\ -\ residential\ only)$$

Where:

ED = Exposure Duration (years)
CF = Conversion Factor (1g/1,000 mg)
EF = Exposure Frequency (days/yr)
IR = Intake Rate (mg/day)
IF = Ingestion Factor (mg-yr/day)

4.0 TOXICITY ASSESSMENT

This section provides toxicological information on the COPCs at the Monsanto Plant. Quantitative estimates of toxic response developed by EPA (1991a) are used to evaluate potential cancer and non-cancer toxicity of the COPCs. Generally, cancer risks are calculated using toxicity factors known as slope factors (SFs), while noncancer hazards are estimated using reference doses (RfDs).

SFs have been developed by EPA for estimating excess lifetime cancer risks associated with exposure to potential carcinogens. For example, SFs for non-radionuclides are expressed in units of $(mg/kg\text{-}day)^{-1}$ and are multiplied by the estimated intake in mg/kg-day, to provide an upper-bound estimate of risk associated with exposure at that intake level. Radionuclide SFs are expressed in terms of activity instead of mass, and are based on best mean estimates of risks rather than upper bound estimates. This approach lessens the potential of overestimating cancer risks. SFs are derived from the results of human epidemiological studies or chronic animal bioassay data, to which mathematical extrapolations from high to low dose, and from animal to human doses, have been applied. The SFs used in this risk assessment are provided in Appendix A.

RfDs have been developed by EPA for indicating the potential for adverse health effects from exposure to chemicals exhibiting noncarcinogenic effects. The RfD is an estimate of lifetime daily exposure for humans (including sensitive subpopulations) which is not likely to cause adverse hazardous effects. RfDs for ingestion exposures are expressed in units of mg/kg-day. Estimated intakes of COPCs from environmental media (e.g., the amount of a COPC ingested from contaminated drinking water) are compared to the RfD. The RfD for each chemical is derived from human epidemiological studies or animal studies to which uncertainty factors have been applied. To understand site hazards, toxicological uncertainty factors should be reviewed for each chemical evaluated. Table 4-1 lists uncertainty factors and critical effects for each RfD. These factors and effects were obtained from the EPA Integrated Risk Information System (IRIS), or if no IRIS values were available, from the Health Effects Assessment Summary Tables (HEAST), and EPA Environmental Criteria and Assessment Office (ECAO) memoranda, as noted.

Table 4-2 presents the SFs that were used to calculate potential cancer risks associated with the carcinogenic COPCs.

Table 4-1
COPC Reference Doses and Uncertainty Factors Used in Calculating Noncarcinogenic Hazards

COPC	Oral RfD (mg/kg-day)	Uncertainty Factor	Critical Effect	Source
Arsenic	3.0E-04	3	Skin Keratosis/hyperpigmentation	2
Beryllium	5.0E-03	100	NOAEL	1
Cadmium (water)	5.0E-04	10	Renal Proteinuria	1
Cadmium (dietary)	1.0E-03	10	Renal Proteinuria	1
Fluoride	6.0E-02	1	Cosmetic dental fluorosis	1
Manganese (water)	5.0E-03	1	Neurological	1
Molybdenum	5.0E-03	30	Respiratory; Renal	1
Nitrate as N	1.6E+00	1	Increased Methemoglobin	1
Selenium	5.0E-03	3	Clinical selenosis	1
Vanadium	7.0E-03	100	NOAEL	2

Source 1 = IRIS Database, 5/94
Source 2 = HEAST Tables, 3/92, 3/93

Table 4-2
COPC Slope Factors Used in Calculating Carcinogenic Risks

COPC	Oral	Inhalation	Carcinogenic Classification	Cancer Type
Arsenic	1.75[a]	50	A	Skin
Beryllium	4.3	8.4	B2	Lung
Cadmium	NA	6.1	B1	Lung
RADIONUCLIDES	**Oral**	**Inhalation**	**External**	
Lead-210+D	6.6E-10	4.0E-9	1.6E-10	
Radium-226+D	1.2E-10	3.0E-9	6.0E-6	
Thorium-230	1.3E-11	2.9E-8	5.4E-11	
Uranium-238+D	2.0E-11	2.4E-8	5.1E-8	

Source: HEAST 3/92, 3/93 and IRIS 5/94.

a = Derived from a unit of risk of 5E-5 μg/l in drinking water.

A = Human Carcinogen - human evidence confirms
B1 = Probable Human Carcinogen - limited human evidence
B2 = Probable Human Carcinogen - sufficient animal evidence

4.1 CHEMICAL TOXICITY

Two oral RfDs are available for cadmium, depending on the route of exposure. Surface-soil toxicity was based on the RfD derived from dietary studies, whereas toxicity associated with groundwater ingestion was evaluated using the RfD based on drinking water studies.

The chemical carcinogens listed in Table 4-2 (arsenic, beryllium, and cadmium) are classified based on their extent of producing cancers in animals and humans (i.e., their weight-of-evidence classification). Arsenic is a class A carcinogen based on sufficient human evidence. Beryllium is classified as a probable human carcinogen (class B2) based on sufficient evidence in animals; and cadmium is classified as a probable human carcinogen (class B1) based on limited human evidence.

Toxicity profiles for the contaminants of concern, as defined in Section 5, are provided in Appendix D. The toxicity profiles provide detailed information on the derivation of the various SFs and RfDs; the types of cancer or noncancer effects; and, the levels of confidence or uncertainties associated with the toxicological data.

4.2 RADIONUCLIDE TOXICITY

The most common pathologies associated with radionuclide exposures are various types of cancer. The following sections explain the methods used to determine cancer risk and briefly describe the mechanism of carcinogenicity.

4.2.1 Radionuclide Slope Factors

HEAST (USEPA 1994c) lists SFs for calculating lifetime cancer risks resulting from exposure to radionuclides. These SFs take into account the biological damage resulting from alpha and beta particle deposition as well as gamma irradiation.

The slope factor methodology for radionuclides, used in this risk analysis, follows guidance as outlined in Exhibit 1 of HEAST. The decay products (+D) of parent isotopes contribute to a major portion of summary cancer risk. Thus, the +D SFs for ingestion, inhalation, and external exposures are used to determine total cancer risk.

Data were available for measured activity levels of lead-210, radium-226, and uranium-238; therefore, total risks result from the sum of component risks for each of these isotopes using the specific +D SFs for each isotope.

4.2.2 Mechanisms of Radionuclide Toxicity

Radionuclides are a human health concern because of the potential carcinogenicity of alpha, beta, and gamma particles. Radionuclides emit ionizing radiation, a form of radiant energy. This energy can remove or add electrons (ionization) to any molecule in its path. The resultant effect is the disruption of DNA, cellular proteins, and human biological processes. The radioactive isotopes in this risk assessment are of concern, primarily because of their alpha, beta, and gamma

emission energies. Risk from alpha and beta particles is determined primarily from use of the ingestion and inhalation +D SFs. In contrast, cancer risks resulting from exposure to gamma emissions are measured primarily by external SFs.

Many of the decay products of uranium-238, radium-226, and lead-210 are of concern because of their alpha particle emissions. These radionuclides can be ingested or inhaled as particulates resulting in tissue deposition. Radiation during initial deposition, and throughout radioactive decay inside the body, can affect cellular function and structure. Radon and its decay products are also of concern because the inhalation of the decay products results in irradiation of sensitive airways.

4.3 ADDITIVITY OF HAZARDS

When assessing cumulative effects of chemical specific hazards, similarity among pathologies enables a summation of hazard quotients (if RfDs are available) to yield a hazard index. Similar pathologies are generally determined by effect types (either critical or secondary) and target organs or systems. With a few minor exceptions, hazards posed by COPCs listed in Section 2 generally do not contain similarities in toxic endpoints. However, arsenic and vanadium hazards may be added in the exposure pathways, due to similarities in hemopoietic and pulmonary effects.

5.0 RISK CHARACTERIZATION

Risk characterization involves estimating the magnitude of the potential adverse health effects of the chemicals under study and making summary judgments about the nature of the potential health threat to the public. Risk characterization combines the results of the dose-response (toxicity) and exposure assessments along with an uncertainty analysis to provide estimates of health risks. In this chapter, risks for all current and future scenarios are characterized, each COPC is evaluated to identify COCs, and uncertainties are discussed. It is common practice to consider risk characterization separately for carcinogenic and noncarcinogenic effects. This is because of a fundamental difference in the way organisms typically respond following exposure to carcinogenic or noncarcinogenic agents. For assessing noncarcinogenic effects, a threshold of exposure is assumed to exist below which there is only a very small likelihood of adverse health impacts in an exposed individual. Exposure to carcinogenic compounds, however, is not thought to be characterized by the existence of a threshold. Rather, all levels of exposure are considered to carry a finite risk of adverse effects.

5.1 RISK CHARACTERIZATION METHODOLOGY

Incremental cancer risk from chemical contaminants is evaluated by combining exposure information with chemical-specific cancer potency information. For the Monsanto site risk assessment, estimated exposure in terms of daily intake averaged over a lifetime (calculated in Section 3) were combined with cancer SFs (presented in Section 4).

In most cases, chemical SFs have been developed based on data derived from animal experiments. When working from animal data, EPA calculates the upper-bound cancer potency based on high-dose to low-dose extrapolation with the intention that risks to humans will not be under-estimated. Thus chemical SFs represent the plausible upper-bound estimate of the excess risk per unit exposure; true risks are likely to be lower. Cancer risk from exposure to multiple chemicals and multiple pathways are treated as additive.

For radionuclides, EPA's SFs are based on human dose-response data. Radiogenic cancers in humans have been observed, for the most part, only following doses of ionizing radiation that are relatively high compared to those likely to result from a combination of background radiation and environmental contaminant radiation. Therefore, a dose-response model must be chosen to allow extrapolation from the number of radiogenic cancers observed at high doses to the number predicted at low doses from all causes including background radiation. EPA's estimates of radiation risks are based on a presumed linear dose-response function.

Radionuclide SFs are characterized as best estimates (i.e., median or 50th percentile values) of the age-averaged lifetime total excess cancer risk per unit intake or exposure. Cancer risks from exposure to multiple radionuclides and multiple radionuclide exposure pathways are assumed to be additive.

The procedure for calculating risk associated with exposure to carcinogenic compounds has been established by EPA (USEPA 1989a). To derive an estimate of risk, the media concentration is

multiplied by the cancer slope factor and estimated intake (or exposure) factor:

$$Risk = C \times IF \times SF$$

where:

Risk = Estimate of the excess lifetime cancer risk to an individual (unitless probability)
C = Concentration (or activity) of chemical in a particular environmental medium
IF = Chronic daily intake or exposure factor, averaged over a 70 year period
SF = Estimate of the slope of the dose response curve (50% or 95% UCL)

Carcinogenic risks are therefore estimated as the incremental probability of an individual developing cancer over a lifetime as a result of exposure to a carcinogen.

Potential effects resulting from chronic exposure to non-carcinogenic compounds are assessed by dividing exposure levels (dose estimates) by chronic RfDs to calculate a hazard quotient (HQ) for each COPC. RfDs represent a dose which would not be expected to result in adverse effects in a sensitive individual.

$$HQ = \frac{Dose}{RfD}$$

where:

HQ = Hazard Quotient: potential for adverse noncarcinogenic effects
Dose = Average daily dose for chronic exposure (mg/kg-day) and
RfD = Acceptable intake for chronic exposure (mg/kg-day)

A hazard quotient less than 1 indicates that the exposure is below the reference dose, and no adverse effects are expected. HQ values do not represent a probability or percentage of occurrence of adverse effects. Because RfDs incorporate a margin of safety, in many cases the likelihood of adverse effects remains very small even at HI above 1.

5.2 CONTAMINANTS OF CONCERN

The COPCs reported in Section 2 represent chemicals that exceeded both background and risk-based screening concentrations. COCs were identified from the list of COPCs to further focus the risk assessment on those chemicals that pose the greatest potential risks to human health.

Selection of COCs is based on the following two criteria:

- A chemical-specific RME cancer risk greater than the corresponding chemical-specific background risk, and greater than 1E-6; or,
- An HQ of 1 or greater for non-carcinogenic effects.

5.3 ESTIMATES OF HAZARDS AND RISKS IN THE INDUSTRIAL SCENARIOS

This section presents risks to workers from exposure to contaminants in materials released from the site. Current scenarios represent an estimate of risks from exposures occurring during current facility activities. During current operations, workers contact contaminated waste materials relatively infrequently. Future scenarios represent risks to workers if site use changes and future workers are exposed to contaminated materials for longer exposure durations.

Descriptions of the industrial scenarios and associated EPCs were presented in Sections 3.3 and 3.4, respectively. Source material ingestion, dust inhalation, and external exposures were evaluated in the current and future industrial scenarios. Appendix B provides the detailed calculations of hazards and risks for each COPC in each pathway for each scenario. Only those chemicals that met the above criteria for a COC were evaluated further in the risk characterization.

Because several of the COCs may exhibit carcinogenic risks at or near background concentrations, a presentation of risk at background was also calculated for each scenario.

5.3.1 Current Industrial Risks

The current industrial risk estimates are summarized in Tables 5-1a to 5-f. All calculated HQs were less than one; therefore no non-carcinogenic COCs were identified. Only carcinogenic risks from exposure to chemicals and radionuclides at the source areas were evident. The chemical COCs were arsenic, beryllium, and cadmium. The radionuclide COCs were lead-210+D, radium-226+D, thorium-230, and uranium-238+D.

Total excess lifetime cancer risks associated with metals ranged from 1E-6 (at the Treater Dust area) to 3E-5 (at the Underflow Solids area). Generally, inhalation and ingestion of arsenic were the principal contributors to the total risk estimates. External exposure to radium-226+D dominated the total excess lifetime cancer risks associated with radionuclides; the total radionuclide risk estimates ranged from 7E-5 (at the Treater Dust area) to 5E-4 (at the Slag area).

Baghouse Dust Area. The reclaim operator was assumed to be working outdoors (unshielded) at the baghouse dust area for 1.5 hours per day, 250 days per year, for 25 years. The total excess lifetime cancer risks associated with potential exposure to metals was 5E-6, predominantly reflecting ingestion of arsenic (3E-6). External exposure to radium-226+D dominated the excess lifetime cancer risk estimate of 2E-4 associated with potential radionuclide exposure.

Nodules Area. The bin operator was assumed to be working outdoors (unshielded) at the nodules area for 1.5 hours per day, 250 days per year, for 25 years. The total excess lifetime cancer risks associated with potential exposure to metals was 3E-6, predominantly reflecting ingestion of beryllium (1E-6), and inhalation of arsenic (7E-7) and cadmium (8E-7). External exposure to radium-226+D dominated the excess lifetime cancer risk estimate of 3E-4 associated with potential radionuclide exposure.

Table 5-1a
Current Industrial Scenario
Risks at Baghouse Dust Area

Contaminant of Concern	Cancer Risks			
	Ingestion	External	Inhalation	TOTAL RISK
Arsenic	3E-6	--	7E-7	3E-6
Beryllium	4E-7	--	2E-8	4E-7
Cadmium	--	--	8E-7	8E-7
Pathway Risk	3E-6	--	2E-6	5E-6
RADIONUCLIDES				
Lead-210+D	2E-6	2E-8	2E-7	2E-6
Radium-226+D	1E-7	2E-4	8E-8	2E-4
Thorium-230	1E-8	2E-9	8E-7	8E-7
Uranium-238+D	2E-8	2E-6	6E-7	3E-6
Pathway Risk	2E-6	2E-4	2E-6	2E-4

Shading indicates Total Risk from all chemicals and pathways.

Table 5-1b
Current Industrial Scenario
Risks at Nodules Area

Contaminant of Concern	Cancer Risks			
	Ingestion	External	Inhalation	TOTAL RISK
Arsenic	4E-7	--	7E-7	1E-6
Beryllium	1E-6	--	2E-8	1E-6
Cadmium	--	--	8E-7	8E-7
Pathway Risk	1E-6	--	2E-6	3E-6
RADIONUCLIDES				
Lead-210+D	--	--	2E-7	2E-7
Radium-226+D	2E-7	3E-4	8E-8	3E-4
Thorium-230	2E-8	3E-9	8E-7	8E-7
Uranium-238+D	3E-8	3E-6	6E-7	3E-6
Pathway Risk	3E-7	3E-4	2E-6	3E-4

Shading indicates Total Risk from all chemicals and pathways.

Table 5-1c
Current Industrial Scenario
Risks at Slag Area

Contaminant of Concern	Cancer Risks			
	Ingestion	External	Inhalation	TOTAL RISK
Arsenic	6E-7	--	8E-7	1E-6
Beryllium	2E-6	--	2E-8	2E-6
Cadmium	--	--	1E-6	1E-6
Pathway Risk	3E-6	--	2E-6	5E-6
RADIONUCLIDES				
Lead-210+D	4E-7	2E-9	3E-7	7E-7
Radium-226+D	6E-7	5E-4	9E-8	5E-4
Thorium-230	6E-8	4E-9	1E-6	1E-6
Uranium-238+D	8E-8	4E-6	7E-7	4E-6
Pathway Risk	1E-6	5E-4	2E-6	5E-4

Shading indicates Total Risk from all chemicals and pathways.

Table 5-1d
Current Industrial Scenario
Risks at Road Dust Area

Contaminant of Concern	Cancer Risks			
	Ingestion	External	Inhalation	TOTAL RISK
Arsenic	4E-6	--	2E-6	5E-6
Beryllium	8E-7	--	4E-8	8E-7
Cadmium	--	--	2E-6	2E-6
Pathway Risk	5E-6	--	4E-6	8E-6
RADIONUCLIDES				
Lead-210+D	1E-5	5E-8	6E-7	1E-5
Radium-226+D	5E-7	4E-4	2E-7	4E-4
Thorium-230	5E-8	4E-9	2E-6	2E-6
Uranium-238+D	8E-8	3E-6	1E-6	5E-6
Pathway Risk	1E-5	4E-4	4E-6	4E-4

Shading indicates Total Risk from all chemicals and pathways.

Table 5-1e
Current Industrial Scenario
Risks at Treater Dust Area

Contaminant of Concern	Cancer Risks			
	Ingestion	External	Inhalation	TOTAL RISK
Arsenic	--	--	4E-7	4E-7
Beryllium	3E-7	--	1E-8	3E-7
Cadmium	--	--	5E-7	5E-7
Pathway Risk	3E-7	--	1E-6	1E-6
RADIONUCLIDES				
Lead-210+D	6E-6	2E-8	1E-7	6E-6
Radium-226+D	8E-8	6E-5	5E-8	6E-5
Thorium-230	8E-9	6E-10	5E-7	5E-7
Uranium-238+D	1E-8	5E-7	4E-7	9E-7
Pathway Risk	6E-6	6E-5	1E-6	7E-5

Shading indicates Total Risk from all chemicals and pathways.

Table 5-1f
Current Industrial Scenario
Risks at Underflow Solids Area

Contaminant of Concern	Cancer Risks			
	Ingestion	External	Inhalation	TOTAL RISK
Arsenic	2E-5	--	2E-6	3E-5
Beryllium	4E-6	--	5E-8	4E-6
Cadmium	--	--	2E-6	2E-6
Pathway Risk	3E-5	--	4E-6	3E-5
RADIONUCLIDES				
Lead-210+D	2E-5	9E-8	6E-7	2E-5
Radium-226+D	5E-7	4E-4	2E-7	4E-4
Thorium-230	6E-8	4E-9	2E-6	2E-6
Uranium-238+D	9E-8	4E-6	2E-6	5E-6
Pathway Risk	2E-5	4E-4	5E-6	4E-4

Shading indicates Total Risk from all chemicals and pathways.

Slag Area. The pot carrier operator was assumed to be potentially exposed to COCs associated with the slag pile while working in heavy equipment for 4 hours per day, 250 days per year, for 25 years. The total excess lifetime cancer risks associated with potential exposure to metals was 5E-6, predominantly reflecting ingestion of beryllium (2E-6), and inhalation of arsenic (8E-7) and cadmium (1E-6). External exposure to radium-226+D dominated the excess lifetime cancer risk estimate of 5E-4 associated with potential radionuclide exposure.

Road Dusts Area. The water truck operator was assumed to be potentially exposed to COCs associated with road dusts while working in heavy equipment for 6 hours per day, 160 days per year, for 25 years. The total excess lifetime cancer risks associated with potential exposure to metals was 8E-6, predominantly reflecting ingestion (4E-6) and inhalation (2E-6) of arsenic, and inhalation of cadmium (2E-6). External exposure to radium-226+D dominated the excess lifetime cancer risk estimate of 4E-4 associated with potential radionuclide exposure.

Treater Dust Area. The loader operator was assumed to be potentially exposed to COCs associated with treater dust while working in heavy equipment for 1 hour per day, 250 days per year, for 25 years. The total excess lifetime cancer risks associated with potential exposure to metals was 1E-6, predominantly reflecting inhalation of arsenic (4E-7) and cadmium (5E-7). External exposure to radium-226+D dominated the excess lifetime cancer risk estimate of 7E-5 associated with potential radionuclide exposure.

Underflow Solids Area. The loader operator was assumed to be potentially exposed to COCs associated with underflow solids while working in heavy equipment for 6 hours per day, 180 days per year, for 25 years. The total excess lifetime cancer risks associated with potential exposure to metals was 3E-5, predominantly reflecting ingestion of arsenic (2E-5). External exposure to radium-226+D dominated the excess lifetime cancer risk estimate of 4E-4 associated with potential radionuclide exposure.

5.3.2 Future Industrial Risks

For the purposes of the future industrial scenario, it was assumed that the Monsanto facility would close down without any site cleanup and alternate industrial activity would occur on-site. This plant-shutdown scenario was developed solely for the purposes of generating health-protective estimates of potential future exposure and risk. The Monsanto facility is likely to continue operations for at least another 20 years and would probably remediate any process-related waste material prior to site abandonment.

EPA, Region 10 default exposure assumptions were used to derive future industrial risk estimates. It was assumed that an individual would be potentially exposed to COCs within a single source area for 8 hours per day, 250 days per year, for 25 years. The total excess lifetime cancer risks associated with the future industrial scenario are presented in Tables 5-2a to 5-2f. Details of the future industrial risk calculations are presented in Appendix B, Table B-2a.

The future industrial inhalation risk estimates were based on emissions originating only from wind-blown erosion of stockpiled material; process-related emissions were not included in the total PM-10 estimate.

Total excess lifetime cancer risks associated with metals ranged from 3E-6 (at the Treater Dust area) to 5E-5 (at the Underflow Solids area). Generally, ingestion of arsenic and beryllium were the principal contributors to the total risk estimates. External exposure to radium-226+D dominated the total excess lifetime cancer risks associated with radionuclides; the total radionuclide risk estimates ranged from 1E-3 (at the Baghouse Dust and Treater Dust areas) to 2E-3 at all other areas.

Baghouse Dust Area. The total excess lifetime cancer risks associated with potential exposure to metals was 2E-5, predominantly reflecting ingestion of arsenic (2E-5). External exposure to radium-226+D dominated the excess lifetime cancer risk estimate of 1E-3 associated with potential radionuclide exposure.

Nodules Area. The total excess lifetime cancer risks associated with potential exposure to metals was 9E-6, predominantly reflecting ingestion of beryllium (6E-6) and arsenic (2E-6). External exposure to radium-226+D dominated the excess lifetime cancer risk estimate of 2E-3 associated with potential radionuclide exposure.

Slag Area. The total excess lifetime cancer risks associated with potential exposure to metals was 6E-6, predominantly reflecting ingestion of beryllium (4E-6) and arsenic (1E-6). External exposure to radium-226+D dominated the excess lifetime cancer risk estimate of 2E-3 associated with potential radionuclide exposure.

Road Dust Area. The total excess lifetime cancer risks associated with potential exposure to metals was 1E-5, predominantly reflecting ingestion of beryllium (2E-6) and arsenic (8E-6). External exposure to radium-226+D dominated the excess lifetime cancer risk estimate of 2E-3 associated with potential radionuclide exposure.

Treater Dust Area. The total excess lifetime cancer risks associated with potential exposure to metals was 3E-6, predominantly reflecting ingestion of beryllium (3E-6). External exposure to radium-226+D dominated the excess lifetime cancer risk estimate of 1E-3 associated with potential radionuclide exposure.

Underflow Solids Area. The total excess lifetime cancer risks associated with potential exposure to metals was 5E-5, predominantly reflecting ingestion of arsenic (4E-5). External exposure to radium-226+D dominated the excess lifetime cancer risk estimate of 1E-3 associated with potential radionuclide exposure.

5.3.3 Background Risks - Industrial

The concentrations of several site-related COCs in background soil samples yielded excess lifetime cancer risks ranging from 2E-7 to 7E-5. Consequently, it is important to compare site-related risks with those associated with background to provide an estimate of the magnitude of risks over background. The calculation of background risks for specific COCs in the industrial scenarios is presented in Tables C-1 and C-2 of Appendix C. Background soil data were used to calculate EPCs as described in Section 3.3.1. The industrial exposure assumptions were identical for calculating risks at background and on-site.

Table 5-2a
Future Industrial Scenario
Risks at Baghouse Dust Area

Contaminant of Concern	Cancer Risks			
	Ingestion	External	Inhalation	TOTAL RISK
Arsenic	2E-5	--	6E-7	2E-5
Beryllium	2E-6	--	5E-8	2E-6
Cadmium	--	--	3E-8	3E-8
Pathway Risk	2E-5	--	7E-7	2E-5
RADIONUCLIDES				
Lead-210+D	1E-5	1E-7	8E-7	1E-5
Radium-226+D	7E-7	1E-3	1E-7	1E-3
Thorium-230	8E-8	1E-8	1E-6	1E-6
Uranium-238+D	1E-7	1E-5	1E-6	1E-5
Pathway Risk	1E-5	1E-3	3E-6	1E-3

Shading indicates Total Risk from all chemicals and pathways.

Table 5-2b
Future Industrial Scenario
Risks at Nodules Area

Contaminant of Concern	Cancer Risks			
	Ingestion	External	Inhalation	TOTAL RISK
Arsenic	2E-6	--	6E-7	3E-6
Beryllium	6E-6	--	5E-8	6E-6
Cadmium	--	--	3E-8	3E-8
Pathway Risk	8E-6	--	7E-7	9E-6
RADIONUCLIDES				
Lead-210+D	--	--	8E-7	8E-7
Radium-226+D	1E-6	2E-3	1E-7	2E-3
Thorium-230	1E-7	2E-8	1E-6	1E-6
Uranium-238+D	2E-7	1E-5	1E-6	2E-5
Pathway Risk	1E-6	2E-3	3E-6	2E-3

Shading indicates Total Risk from all chemicals and pathways.

Table 5-2c
Future Industrial Scenario
Risks at Slag Area

Contaminant of Concern	Cancer Risks			
	Ingestion	External	Inhalation	TOTAL RISK
Arsenic	1E-6	--	6E-7	2E-6
Beryllium	4E-6	--	5E-8	5E-6
Cadmium	--	--	3E-8	3E-8
Pathway Risk	6E-6	--	7E-7	6E-6
RADIONUCLIDES				
Lead-210+D	8E-7	6E-9	8E-7	2E-6
Radium-226+D	1E-6	2E-3	1E-7	2E-3
Thorium-230	1E-7	2E-8	1E-6	1E-6
Uranium-238+D	2E-7	1E-5	1E-6	1E-5
Pathway Risk	2E-6	2E-3	3E-6	2E-3

Shading indicates Total Risk from all chemicals and pathways.

Table 5-2d
Future Industrial Scenario
Risks at Road Dust Area

Contaminant of Concern	Cancer Risks			
	Ingestion	External	Inhalation	TOTAL RISK
Arsenic	8E-6	--	6E-7	8E-6
Beryllium	2E-6	--	5E-8	2E-6
Cadmium	--	--	3E-8	3E-8
Pathway Risk	1E-5	--	7E-7	1E-5
RADIONUCLIDES				
Lead-210+D	2E-5	2E-7	8E-7	2E-5
Radium-226+D	9E-7	2E-3	1E-7	2E-3
Thorium-230	1E-7	1E-8	1E-6	1E-6
Uranium-238+D	2E-7	1E-5	1E-6	1E-5
Pathway Risk	2E-5	2E-3	3E-6	2E-3

Shading indicates Total Risk from all chemicals and pathways.

Table 5-2e
Future Industrial Scenario
Risks at Treater Dust Area

Contaminant of Concern	Cancer Risks			
	Ingestion	External	Inhalation	TOTAL RISK
Arsenic	--	--	6E-7	6E-7
Beryllium	3E-6	--	5E-8	3E-6
Cadmium	--	--	3E-8	3E-8
Pathway Risk	3E-6	--	7E-7	3E-6
RADIONUCLIDES				
Lead-210+D	4E-5	3E-7	8E-7	5E-5
Radium-226+D	6E-7	1E-3	1E-7	1E-3
Thorium-230	7E-8	9E-9	1E-6	1E-6
Uranium-238+D	9E-8	7E-6	1E-6	8E-6
Pathway Risk	4E-5	1E-3	3E-6	1E-3

Shading indicates Total Risk from all chemicals and pathways.

Table 5-2f
Future Industrial Scenario
Risks at Underflow Solids Area

Contaminant of Concern	Cancer Risks			
	Ingestion	External	Inhalation	TOTAL RISK
Arsenic	4E-5	--	6E-7	5E-5
Beryllium	7E-6	--	5E-8	7E-6
Cadmium	--	--	3E-8	3E-8
Pathway Risk	5E-5	--	7E-7	5E-5
RADIONUCLIDES				
Lead-210+D	4E-5	3E-7	8E-7	4E-5
Radium-226+D	9E-7	1E-3	1E-7	1E-3
Thorium-230	1E-7	1E-8	1E-6	1E-6
Uranium-238+D	2E-7	1E-5	1E-6	1E-5
Pathway Risk	4E-5	1E-3	3E-6	2E-3

Shading indicates Total Risk from all chemicals and pathways.

The comparison of site risks to background is presented in Table 5-3, in addition to the calculation of incremental risks estimates, which were derived by subtracting the background risk from the site risk. In general, the background risks were approximately one order of magnitude lower than the site risks. Consequently, subtracting the background risk estimate yielded an incremental risk that was similar to the site risk.

5.4 ESTIMATES OF HAZARDS AND RISKS IN THE RESIDENTIAL SCENARIOS

This section presents risks to residents from exposure to contaminants in materials released from the site. Current scenarios represent an estimate of risks from exposures to contaminated soil, process-related air emissions, and windblown dust. Future scenarios represent risks to residents from exposure to soil and dust, and for residents to the south of the facility, from potential use of groundwater as drinking water.

As stated in Section 3, exposure to surface soils was evaluated for non-cancer hazards and cancer risks in adult and child residents (an integrated approach). Inhalation of respirable particulates, ingestion of soils, ingestion of ground water, and external exposure to radionuclides were evaluated as pathways for the residential scenario. The exposure and toxicity assessments were combined to present conservative, upper-bound estimates of hazards and lifetime cancer risks.

5.4.1 Current Residential Risks

Current residential risks were based on the EPA default exposure parameters as presented in Section 3.3.3, which assumed that an individual is exposed to the contaminated media 350 days/year for 30 years under the RME scenario. The estimated hazards and excess lifetime cancer risks presented below only pertain to the exposure assumptions, and do not represent actual risks to existing residents.

All calculated HQs associated with ingestion of soils were less than one; therefore, no non-carcinogenic COCs were identified in soils. Only carcinogenic risks from exposure to chemicals and radionuclides in soil and airborne particulates were evident. The chemical COCs for the residential scenario were arsenic, beryllium, and cadmium. The radionuclide COCs were lead-210+D, radium-226+D, thorium-230, and uranium-238+D.

Tables 5-4a through 5-4c present the RME and average hazards and risks from COCs associated with the current residential scenarios. Appendix B, Tables B-4a through B-4c show all RME and average risk calculations by pathway.

In general, the total estimated RME excess lifetime cancer risks from the chemical COCs ranged from 1E-5 to 3E-5, with most of the risks associated with the ingestion pathway. The average risks were approximately one order of magnitude lower.

Excess lifetime cancer risks from radionuclides ranged from 4E-6 to 1E-5 with distinct pathway risks for each residence.

Table 5-3
Incremental Risk Over Background
Industrial Scenarios

	CURRENT SCENARIO RISK			FUTURE SCENARIO RISK		
	Site[a]	Background[b]	Increment over Background	Site[a]	Background[b]	Increment over Background
BAGHOUSE DUST AREA						
Metals	5E-6	3E-7	4E-6	2E-5	1E-6	2E-5
Radionuclides	2E-4	1E-5	2E-4	1E-3	7E-5	1E-3
NODULES AREA						
Metals	3E-6	3E-7	3E-6	9E-6	1E-6	8E-6
Radionuclides	3E-4	1E-5	3E-4	2E-3	7E-5	2E-3
SLAG AREA						
Metals	5E-6	7E-7	4E-6	6E-6	1E-6	5E-6
Radionuclides	5E-4	2E-5	5E-4	2E-3	7E-5	2E-3
ROAD DUSTS AREA						
Metals	8E-6	6E-7	8E-6	1E-5	1E-6	9E-6
Radionuclides	4E-4	2E-5	4E-4	2E-3	7E-5	1E-3
TREATER DUST AREA						
Metals	1E-6	2E-7	1E-6	3E-6	1E-6	2E-6
Radionuclides	7E-5	5E-6	7E-5	1E-3	7E-5	1E-3
UNDERFLOW SOLIDS AREA						
Metals	3E-5	7E-7	3E-5	5E-5	1E-6	5E-5
Radionuclides	4E-4	2E-5	4E-4	2E-3	7E-5	1E-3

(a) Includes ingestion, external, and inhalation.
(b) Includes ingestion and external.

Table 5-4a
Current Risks at Southern Residence

Contaminant of Concern	RME Risks				AVG Risks
	Ingestion	External	Inhalation	Total	Total
Arsenic	2E-5	--	1E-6	3E-5	2E-6
Beryllium	--	--	7E-8	7E-8	2E-8
Cadmium	--	--	2E-6	2E-6	6E-7
Pathway Risk	2E-5	--	4E-6	3E-5	2E-6
RADIONUCLIDES					
Lead-210+D	2E-6	1E-8	6E-7	3E-6	7E-7
Radium-226+D	--	--	4E-7	4E-7	9E-8
Thorium-230	3E-8	3E-9	4E-6	4E-6	9E-7
Uranium-238+D	5E-8	3E-6	3E-6	6E-6	1E-6
Pathway Risk	2E-6	3E-6	8E-6	1E-5	3E-6

Shading indicates Total Risk from all chemicals and pathways.

Table 5-4b
Current Risks at Western Residence

Contaminant of Concern	RME Risks				AVG Risks
	Ingestion	External	Inhalation	Total	Total
Arsenic	--	--	4E-7	4E-7	1E-7
Beryllium	1E-5	--	2E-8	1E-5	4E-9
Cadmium	--	--	7E-7	7E-7	2E-7
Pathway Risk	1E-5	--	1E-6	1E-5	3E-7
RADIONUCLIDES					
Lead-210+D	5E-6	3E-8	2E-7	6E-6	9E-7
Radium-226+D	--	--	7E-8	7E-8	2E-8
Thorium-230	3E-8	2E-9	7E-7	7E-7	2E-7
Uranium-238+D	--	--	5E-7	5E-7	1E-7
Pathway Risk	6E-6	3E-8	1E-6	7E-6	1E-6

Shading indicates Total Risk from all chemicals and pathways.

Table 5-4c
Current Risks at Northern Residence

Contaminant of Concern	RME Risks				AVG Risks
	Ingestion	External	Inhalation	Total	Total
Arsenic	--	--	5E-7	5E-7	1E-7
Beryllium	1E-5	--	1E-8	1E-5	9E-7
Cadmium	--	--	1E-6	1E-6	2E-7
Pathway Risk	1E-5	--	2E-6	1E-5	1E-6
RADIONUCLIDES					
Lead-210+D	3E-6	1E-8	1E-7	3E-6	6E-7
Radium-226+D	--	--	4E-8	4E-8	8E-9
Thorium-230	3E-8	2E-9	4E-7	4E-7	9E-8
Uranium-238+D	--	--	3E-7	3E-7	6E-8
Pathway Risk	3E-6	2E-8	8E-7	4E-6	7E-7

Shading indicates Total Risk from all chemicals and pathways.

Southern Residence. The total RME excess lifetime cancer risk associated with potential exposure to metals was 3E-5; ingestion of arsenic (2E-5) was the primary contributor to the total risk estimate. The total RME excess lifetime cancer risk associated with potential exposure to radionuclides was 1E-5; inhalation of thorium-230 (4E-6) and uranium-238+D (3E-6), in addition to external exposure to uranium-238+D (3E-6) were the primary contributors to the total risk estimate. The total average excess lifetime cancer risk estimate for metals and radionuclides was 2E-6 and 3E-6, respectively.

Western Residence. The total RME excess lifetime cancer risk associated with potential exposure to metals was 1E-5; ingestion of beryllium (1E-5) was the primary contributor to the total risk estimate. The total RME excess lifetime cancer risk associated with potential exposure to radionuclides was 7E-6; ingestion of lead-210+D (5E-6) was the primary contributor to the total risk estimate. The total average excess lifetime cancer risk estimate for metals and radionuclides was 3E-7 and 1E-6, respectively.

Northern Residence. The total RME excess lifetime cancer risk associated with potential exposure to metals was 1E-5; ingestion of beryllium (1E-5) was the primary contributor to the total risk estimate. The total RME excess lifetime cancer risk associated with potential exposure to radionuclides was 4E-6; ingestion of lead-210+D (3E-6) was the primary contributor to the total risk estimate. The total average excess lifetime cancer risk estimate for metals and radionuclides was 1E-6 and 7E-7, respectively.

5.4.2 Future Residential Risks

The exposure assumptions for the hypothetical future scenarios were presented in Section 3.3.4. Location of the future residences were shown on Figure 3-4. Tables 5-5a through 5-5d show the average and RME hazards and risks from the COCs associated with the future residential scenarios. Appendix B, Tables B-4d through B-4g show all risk calculations by pathway.

Southern Residence II. Table 5-5a presents the RME and average hazard quotients and risks associated with the hypothetical future residence located within the residential zoning classification south of the Monsanto property. Hazard quotients of 2 and 1 were associated with ingestion of groundwater containing fluoride and selenium, respectively.

The total RME excess lifetime cancer risk associated with potential exposure to metals was 2E-5; ingestion of arsenic (1E-5) and beryllium (9E-6) was the primary contributor to the total risk estimate. The total RME excess lifetime cancer risk associated with potential exposure to radionuclides was 4E-6; ingestion of lead-210+D (2E-6) was the primary contributor to the total risk estimate.

The total average excess lifetime cancer risk estimate for metals and radionuclides was 1E-6 and 3E-7, respectively.

Northern Residence I. The total RME excess lifetime cancer risk associated with potential exposure to metals was 1E-4; ingestion of arsenic (9E-5) was the primary contributor to the total risk estimate. The total RME excess lifetime cancer risk associated with potential exposure to

Table 5-5a
Future Hazards and Risks at Southern Residence II

Contaminant of Concern	Hazard Quotient[a]	RME Risks				AVG Risks
		Ingestion	External	Inhalation	Total	Total
Arsenic	--	1E-5	--	5E-7	1E-5	1E-7
Beryllium	--	9E-6	--	2E-8	9E-6	8E-7
Cadmium	0.3	--	--	8E-7	8E-7	2E-7
Fluoride	2.3	--	--	--	--	--
Selenium	1.4	--	--	--	--	--
Pathway Risk		2E-5	--	1E-6	2E-5	1E-6
RADIONUCLIDES						
Lead-210+D	--	2E-6	1E-8	2E-7	3E-6	6E-8
Radium-226+D	--	--	--	4E-8	4E-8	1E-8
Thorium-230	--	3E-8	3E-9	4E-7	5E-7	1E-7
Uranium-238+D	--	--	--	3E-7	3E-7	8E-8
Pathway Risk		2E-6	1E-8	1E-6	4E-6	3E-7

Shading indicates Total Risk from all chemicals and pathways.

(a) Hazard from ingestion of groundwater.

Table 5-5b
Future Risks at Northern Residence I

Contaminant of Concern	Hazard Quotient	RME Risks				AVG Risks
		Ingestion	External	Inhalation	Total	Total
Arsenic	--	9E-5	--	5E-6	1E-4	7E-6
Beryllium	--	2E-5	--	1E-7	2E-5	2E-6
Cadmium	--	--	--	8E-6	8E-6	2E-6
Pathway Risk		1E-4	--	1E-5	1E-4	1E-5
RADIONUCLIDES						
Lead-210+D	--	5E-5	2E-7	2E-6	6E-5	1E-5
Radium-226+D	--	2E-6	2E-3	2E-7	2E-3	4E-4
Thorium-230	--	2E-7	2E-8	3E-6	3E-6	7E-7
Uranium-238+D	--	3E-7	1E-5	2E-6	2E-5	4E-6
Pathway Risk		6E-5	2E-3	8E-6	2E-3	4E-4

Shading indicates Total Risk from all chemicals and pathways.

Table 5-5c
Future Risks at Northern Residence II

Contaminant of Concern	Hazard Quotient	RME Risks				AVG Risks
		Ingestion	External	Inhalation	Total	Total
Arsenic	--	3E-5	--	2E-7	3E-5	2E-6
Beryllium	--	9E-6	--	7E-9	9E-6	7E-7
Cadmium	--	--	--	4E-7	4E-7	9E-8
Pathway Risk		4E-5	--	6E-7	4E-5	3E-6
RADIONUCLIDES						
Lead-210+D	--	6E-6	3E-8	1E-7	6E-6	9E-7
Radium-226+D	--	4E-7	4E-4	1E-8	4E-4	8E-5
Thorium-230	--	5E-8	4E-9	2E-7	2E-7	4E-8
Uranium-238+D	--	7E-8	3E-6	1E-7	4E-6	9E-7
Pathway Risk		6E-6	4E-4	4E-7	4E-4	9E-5

Shading indicates Total Risk from all chemicals and pathways.

Table 5-5d
Future Risks at Southern Residence I[a]

Contaminant of Concern	Hazard Quotient	RME Risks				AVG Risks
		Ingestion	External	Inhalation	Total	Total
Arsenic	--	3E-5	--	5E-7	3E-5	2E-6
Beryllium	--	3E-5	--	2E-8	3E-5	2E-6
Cadmium	--	--	--	7E-7	7E-7	2E-7
Pathway Risk		5E-5	--	1E-6	6E-5	4E-6
RADIONUCLIDES						
Lead-210+D	--	2E-5	9E-8	2E-7	2E-5	3E-6
Radium-226+D	--	2E-6	2E-3	4E-8	2E-3	3E-4
Thorium-230	--	3E-7	2E-8	4E-7	7E-7	1E-7
Uranium-238+D	--	3E-7	1E-5	3E-7	1E-5	3E-6
Pathway Risk		2E-5	2E-3	1E-6	2E-3	3E-4

Shading indicates Total Risk from all chemicals and pathways.

(a) Currently unoccupied home.

radionuclides was 2E-3; external exposure to radium-226+D (2E-3) was the primary contributor to the total risk estimate (see Table 5-5b). The total average excess lifetime cancer risk estimate for metals and radionuclides was 1E-5 and 4E-4, respectively.

Northern Residence II. The total RME excess lifetime cancer risk associated with potential exposure to metals was 4E-5; ingestion of arsenic (3E-5) was the primary contributor to the total risk estimate (see Table 5-5c). The total RME excess lifetime cancer risk associated with potential exposure to radionuclides was 4E-4; external exposure to radium-226+D (4E-4) was the primary contributor to the total risk estimate. The total average excess lifetime cancer risk estimate for metals and radionuclides was 3E-6 and 9E-5, respectively.

Southern Residence I. Table 5-5d presents the RME and average risks associated with the hypothetical future residence located at the southern fenceline of the Monsanto facility. Although individuals were in residence at this location until relatively recently, no residents are currently present.

The total RME excess lifetime cancer risk associated with potential exposure to metals was 6E-5; ingestion of arsenic (3E-5) and beryllium (3E-5) was the primary contributor to the total risk estimate. The total RME excess lifetime cancer risk associated with potential exposure to radionuclides was 2E-3; external exposure to radium-226+D (2E-3) was the primary contributor to the total risk estimate. The total average excess lifetime cancer risk estimate for metals and radionuclides was 4E-6 and 3E-4, respectively.

5.4.3 Background - Residential

The concentrations of several site-related COCs in background soil samples yielded excess lifetime cancer risks of 2E-5 to 3E-4 for metals and radionuclides, respectively. Consequently, it is important to compare site-related risks with those associated with background to provide an estimate of the magnitude of risks over background. The calculation of background risks for specific COCs in the residential scenarios is presented in Table C-3 of Appendix C. Background soil data were used to calculate EPCs as described in Section 3.3.1. The residential exposure assumptions were identical for calculating risks at background and residential locations identified in the risk assessment.

The comparison of residential site risks to background is presented in Table 5-6, in addition to the calculation of incremental risks estimates, which were derived by subtracting the background risk from the site risk.

With the exception of the current southern scenario, the current exposure scenarios yielded ingestion and external cancer risks at or below background. The incremental ingestion cancer risk associated with metals at the southern residence was 8E-6. Current inhalation risk estimates ranged from 8E-7 to 8E-6.

Incremental excess lifetime cancer risks associated with ingestion of metals for the future residential scenario ranged from 4E-6 (Southern II) to 1E-4 (Northern I); the radionuclide risks ranged from below background (Southern II) to 2E-3 (Northern I). Future inhalation risk estimates ranged from 4E-7 to 1E-5.

Table 5-6
Incremental Risk Over Background
Residential Scenarios

	SCENARIO RISK			
	Site[a]	Background[b]	Ingest, External	Inhalation
CURRENT SOUTHERN				
Metals	3E-5	2E-5	8E-6	4E-6
Radionuclides	1E-5	3E-4	BB	8E-6
CURRENT WESTERN				
Metals	1E-5	2E-5	BB	1E-6
Radionuclides	7E-6	3E-4	BB	1E-6
CURRENT NORTHERN				
Metals	1E-5	2E-5	BB	2E-6
Radionuclides	4E-6	3E-4	BB	8E-7
FUTURE SOUTHERN II				
Metals	2E-5	2E-5	4E-6	1E-6
Radionuclides	4E-6	3E-4	BB	1E-6
FUTURE NORTHERN I				
Metals	1E-4	2E-5	1E-4	1E-5
Radionuclides	2E-3	3E-4	2E-3	8E-6
FUTURE NORTHERN II				
Metals	4E-5	2E-5	2E-5	6E-7
Radionuclides	4E-4	3E-4	9E-5	4E-7
FUTURE SOUTHERN I[c]				
Metals	6E-5	2E-5	4E-5	1E-6
Radionuclides	2E-3	3E-4	1E-3	1E-6

(a) Includes ingestion, external, and inhalation.
(b) Includes ingestion and external.
(c) Currently unoccupied home.
BB = Risks from COCs in soil below soil background risk.

5.5 RISKS FROM GAMMA IRRADIATION

From the discussions in Sections 5.3 and 5.4, it is evident that the external exposure pathway, primarily from radium-226 and its decay products, contributes substantially to risk in both the industrial and residential scenarios. As discussed in Section 2.9, the gamma measurements from soil samples outside the facility boundary were within the range of background, yet the methodology used to estimate risks suggests that carcinogenic risks from gamma emitting radionuclides (i.e. radium-226 +D in the residential scenarios) are approximately one order of magnitude higher than background risks. This risk is likely overestimated due to the non-uniform distributions of the radionuclide COCs in the 0-1 inch layer of soil. In contrast, exposure to a thick layer of uniformly distributed material would result in greater exposure.

To determine if risks in the industrial scenarios also exceeded background by an order magnitude or more, another methodology was used to compare the estimated risks. All of the gamma measurements recorded at the source-material sampling locations were greater than the calculated background UTL gamma level (Section 2.9). Because the assumptions regarding external exposure principally relate to gamma radiation, risk was also calculated using the methodology discussed in Appendix E.

The Site gamma data collected by MCC include a background component which originates from all gamma emitting sources. Therefore, excess incremental cancer risk was calculated by subtracting the background risk from the Site risk. This result will not contain the contributions from other gamma emitters; the risk will represent the Site contribution.

Table 5-7 compares the external exposure risks under the current industrial scenario (calculated from the activity levels of specific radionuclides, and applying the slope factor approach), to risks calculated using gamma measurements from Appendix E.

Table 5-7
Comparison of Incremental External Exposure Risk Estimates:
Slope Factor Method and Gamma Radiation Measurements[a] for the
Current Industrial Scenarios

Source Area	Slope Factor Method	NESHAPS Approach (Appendix E)
Underflow Solids	4E-4	4E-4
Baghouse Dusts	2E-4	1E-4
Nodules	3E-4	5E-4
Treater Dusts	6E-5	1E-4
Slag - top[b]	5E-4	7E-4

(a) = Risks over background
(b) = Based on gamma readings on top of slag pile rather than slag perimeter

The comparison indicates that the risk estimates are similar (within a factor of 2). These differences are likely to be within the error margin of either methodology. This comparison also suggests that risks from external exposure to large source material piles containing the radionuclide COCs can be approximated using either methodology.

5.6 UNCERTAINTY ANALYSIS

Confidence in this risk assessment depends in part on the accuracy and representativeness of the environmental sampling, exposure assumptions, and toxicological data. Many assumptions of the methodology and approach are inherently or intentionally conservative so that the risk assessment will be more likely to overestimate risk, rather than underestimate it.

5.6.1 Uncertainties Regarding Exposure Factors

Many of the exposure factors used in this risk assessment are default values recommended by EPA. These default factors, which are used nationwide, do not necessarily reflect actual conditions at the site, and are used as assumptions in the absence of site-specific information.

Industrial Exposure Factors - The current RME industrial exposure scenarios developed for this risk assessment were derived from information provided by Monsanto (1993). An evaluation of potential exposures to all individuals working or visiting the plant is beyond the scope of this risk assessment. The RME scenarios only represent a small subset of the existing workforce. As discussed in Section 3.3, it is assumed that individuals working indoors are not exposed to releases from the source areas under consideration.

Because of the dynamic nature of the numerous job tasks at the Monsanto Plant, some individuals may be exposed to more than one source area or may spend a longer time at a particular source area, than what was assumed under the RME scenarios. For example, if an individual operates equipment at the nodules area and also operates equipment at the underflow solids pile, their risks may be underestimated.

Residential Exposure Factors - The residential scenarios were based on EPA default exposure factors as discussed in Section 3.3. These default factors assume that the individuals stay at home for 24 hours/day, 350 days/year, for 30 years. This is likely an overestimation of the amount of time that people are actually at their residences.

Residents may consume garden produce grown in contaminated soil. Due to the lack of site-specific data and the enormous uncertainties associated with determining soil to plant transfer factors, and intake rates of garden fruits, leaves, and roots, this pathway was not quantified. However, an additional incremental risk may occur, assuming consumption of home grown produce in the residential scenarios.

Individuals from these residences or other residences may work in nearby agricultural fields that contain elevated levels of COCs. Quantification of RME exposures (and subsequent risks) from typical agricultural work practices in fields near the Monsanto Plant boundary was not performed

because of large uncertainties (See Section 3.3). It is assumed that an incremental risk will occur from working in contaminated agricultural soils. However, the individual is not expected to receive risks greater than under the conservative residential scenario.

5.6.2 Uncertainties Related to Radionuclide Risks

Exposure to radionuclides depends on several factors and assumptions, each with its own set of uncertainties. Radiation doses to organs and tissues of the body are related to the concentrations of specific radionuclides in environmental media. When radiation originates outside of the body, it is referred to as external exposure. This situation is in contrast to the intake of radionuclides by ingestion or inhalation, where the radiation is emitted from inside the body.

External Dose from Soil - For external exposures, the kinds of radiation of concern are those that can sufficiently penetrate into the body - such as gamma radiation. The radiation dose depends strongly on the temporal and spatial distributions of the radionuclides in the media to which a human is exposed. The calculation of risk from external exposure assumes that any gamma-emitting radionuclide in soil is uniformly distributed in that soil within a finite soil depth and density, and dispersed in an infinite plane geometry.

At the Monsanto Plant, the depositional pattern of radionuclides in soils outside the plant boundary forms a steep concentration gradient outward from the perimeter of the plant. In addition, most of the radionuclides were deposited in a surficial layer (0 to 1 inch depth), resulting in irregular vertical distributions. These non-uniform distributions result in uncertainties that tend to overestimate risks because EPA's risk model assumes an infinitely thick layer of radionuclide-containing material. As noted in Section 2.9 and 5.0, the gamma measurements in soils outside the facility boundary were within the background UCL levels. However, the estimated external risks from the radionuclides using the slope factor method were an order of magnitude greater than background risks. This risk is likely overestimated due to the non-uniform distributions of the COC's in soil. Similarly, the source materials each have varying densities, stratification, and depths. Thus, additional uncertainty is realized when external exposures to source materials are based on the same assumptions regarding soil.

The calculation of external radiation risk also includes two additional factors, the gamma shielding factor (Se) and the gamma exposure time factor (Te) which are adjusted to account for both attenuation of radiation fields due to shielding (e.g., by buildings, terrain, or other barriers) and for exposure times of less than 24 hours per day. In general, the Se and Te assumptions used in this assessment are considered protective.

Ingestion and Inhalation Doses - Specific speciation and solubilities of the radionuclide COCs are unknown. This results in uncertainties regarding their specific gamma and alpha particle irradiation mechanisms to target organs and tissues, which are components of SFs. The mechanisms and contribution of alpha radiation risk relative to gamma radiation risks continues to be a source of scientific debate.

However, the cancer effects related to these two types of radiation are a part of the slope factor uncertainties. In addition, because ingestion and inhalation SFs for radionuclides are based on best estimates of the mean risk per unit intake, some individuals or sub-populations may have

higher sensitivity.

There is uncertainty related to the background component of radiation exposure. As discussed in Section 2.1, measurements of activity (pCi/g) vary depending on such factors as the distribution of radionuclides at each soil sample location and analytical variances. Similarly, background gamma readings could vary considerably depending on instrument accuracy and calibration, and natural variations in ambient background levels.

5.6.3 Uncertainties Involving Exposure and Risks from Radon-222

The inhalation of radon-222 and its decay products is a major dose contributor of internal radiation when radium-226+D is present in the soil. In the BEIR IV report (National Research Council 1988), radon-222 and its decay products were identified as the most important sources of radiation exposure to the general public from naturally occurring radioactivity. Most radon exposure originates from radon concentrated in indoor environments. Typical outdoor concentrations are substantially lower than typical indoor levels. Current methodologies characterize the air exposure pathway by deriving an air/radon concentration based on soil concentrations of radium. The air/soil concentration relationship depends on radon diffusion equations for the soil and for the atmosphere. The radon release rate from soil or source materials varies with the local distribution of radium-226+D, soil type, cover materials, soil density, porosity, moisture content, meteorological factors (e.g., wind speeds and stability classes), decay, and ingrowth. The calculation of outdoor radon concentrations using all of these factors requires a comprehensive atmospheric dispersion model. Such extensive computations are not within the scope of this risk assessment.

Radon gas in the soil may enter buildings by diffusion, or through cracks or holes in foundations by convection. In addition to the above factors affecting radon flux, predictions of indoor radon concentrations also vary with factors such as building characteristics and design, air exchange, and ventilation. The predictability of indoor radon concentrations depends on multi-compartmental and dynamic models with significant amounts of uncertainty.

Nevertheless, risks from radon and its decay products (Rn-222+D) may contribute additional risks where radium-226+D concentrations are elevated over background. The current residences have been occupied for about as long as the plant has been operating. Most of the deposition of radium-226+D is assumed to have occurred adjacent to the residences, and the houses are assumed to not have been constructed on top of soil containing elevated radium concentrations. Some small incremental risks from radon-222+D may occur to these residents; however, such risks are likely to be insignificant relative to existing variations in background radon and uncertainties in calculating either indoor or outdoor concentrations.

5.6.4 Toxicity Values for Radionuclides

Uncertainty is associated with using EPA's radionuclide slope factors to evaluate risks posed to adult workers. The radionuclide slope factors presented in HEAST and used in this assessment are designed to be protective of the general population (i.e., through childhood and adulthood)

and were derived from dosimetric models that account for the age-dependence associated with organ-specific biokinetics and susceptibility to cancer induction. The susceptibility to cancer later in life for children is likely greater than that for adults, reflecting differences in growth rate and metabolism. Consequently, the slope factors presented in HEAST may overestimate risks posed to adult workers.

However, EPA is currently revising its methodology for estimation of cancer risks due to low level radiation exposures (EPA 1994a). Although revised slope factors reflecting this methodology have not yet been published in HEAST (as of January 1995), it is evident that these slope factors will be more conservative than those currently in use. Consequently, the radionuclide risks presented in this assessment represent underestimates relative to the new methodology.

5.7 SUMMARY OF RISKS

5.7.1 Summary of Current Industrial Risks

Most of the excess lifetime cancer risk associated with metals in the current industrial scenario reflects ingestion and inhalation of arsenic. The total incremental lifetime cancer risks ranged from 1E-6 at the Treater Dust area to 3E-5 at the Underflow Solids area.

Concentrations of arsenic, beryllium, and cadmium in on-site source piles exceeded background by 2- to 500-fold. The highest concentrations of arsenic and cadmium were found in the underflow solids, baghouse dusts and road dusts, whereas the highest beryllium concentrations were found in nodules and slag. Fugitive dust emissions within the facility fenceline originated predominantly from the underflow solids.

External exposure to radium-226 dominated the total incremental cancer risks associated with current industrial exposure to radionuclides; the total risk estimates ranged from 7E-5 at the Treater Dust area to 5E-4 at the Slag area. The concentrations (activity levels) of radium-226+D at each source area were approximately 10-fold higher than the background UCL (1.9 pCi/g). The highest radium-226+D activity level (54 pCi/g) was detected in the slag pile.

The gamma radiation measurements and associated risks in the current industrial scenario (as calculated in Appendix E) were consistent with the activity levels and calculated risks using the slope factor methodology.

The Superfund risk assessment process for the evaluation of risks from exposure to uncontrolled releases from hazardous substances in an industrial setting is fundamentally different from methodologies used to derive OSHA standards for the protection of workers. An analysis of the results of this assessment using OSHA requirements is beyond the scope of this document.

5.7.2 Summary of Future Industrial Risks

Most of the excess lifetime cancer risk associated with metals in the future industrial scenario reflects ingestion of arsenic and beryllium. The total incremental lifetime cancer risks ranged

from 2E-6 at the Treater Dust area to 5E-5 at the Underflow Solids area.

External exposure to radium-226 dominated the total incremental cancer risks associated with potential future industrial exposure to radionuclides; the total risk estimates ranged from 1E-3 to 2E-3.

The gamma radiation measurements and associated risks in the future industrial scenario (as calculated in Appendix E) were consistent with the activity levels and calculated risks using the slope factor methodology.

5.7.3 Summary of Current Residential RME Risks

Incremental risks above background for the residential scenarios are presented in Table 5-6. In the current residential scenario, the incremental cancer risk estimate associated with exposure to metals exceeded background only at the southern residence (8E-6). Risks associated with exposure to radionuclides did not exceed background at any location.

5.7.4 Summary of Future Residential RME Risks

In the future residential scenario, the incremental cancer risk estimate associated with exposure to metals ranged from 4E-6 at the future Southern II residence to 1E-4 at the future Northern I residence. Hazard quotients of 2 and 1 were associated with potential future ingestion of groundwater at the Southern II residence.

External exposure to radium-226+D dominated the total incremental cancer risks associated with potential future industrial exposure to radionuclides; the total risk estimates ranged from below background at the future Southern II residence to 2E-3 at the future Northern I residence.Ecology and Environment, Inc., (E & E)

6.0 SUMMARY AND CONCLUSIONS

Human health risks at the Monsanto Plant were evaluated for current and future industrial land use, and current and future residential land use. Arsenic, beryllium, cadmium, lead-210+D, radium-226+D, thorium-230, and uranium-238+D present in soils and source piles were identified as contaminants of concern at the site because they pose potential carcinogenic risks greater than 1-in-1,000,000 (>1E-6). The potential for other, non-cancer health effects was evaluated, but none were found which posed HQs greater than 1. If ground water south of the Plant was used as a drinking water source, exposure to fluoride and selenium would exceed RfDs. The City of Soda Springs drinking water supplies are unaffected by the site.

The findings of the risk assessment support the following conclusions:

CURRENT INDUSTRIAL SCENARIO

The estimates of risk in the current industrial scenario included time and motion data provided by Monsanto to more accurately determine potential exposures.

Potential exposures to chemical carcinogens (arsenic, beryllium, and cadmium) by individuals working on-site yielded incremental lifetime cancer risks ranging from 1E-6 to 3E-5.

Potential exposure to radionuclide COCs (particularly external exposure to radium 226+D) yielded incremental lifetime cancer risk estimates ranging from 7E-5 at the Treater Dust area to 5E-4 at the Slag area. Site risks were approximately 10-fold higher than those associated with background soils. Potential risks associated with external exposure to radium 226+D were generally 100-fold higher than those due to the ingestion or inhalation pathways.

The risk estimates derived from gamma radiation measurements were consistent with those calculated from the activity levels of specific radionuclides.

FUTURE INDUSTRIAL SCENARIO

The estimates of risk in the future industrial scenario included default assumptions employed by EPA, Region 10. This conservative scenario was developed to describe potential future risks in the unlikely event of plant shutdown without remediation.

Potential exposures to chemical carcinogens (arsenic, beryllium, and cadmium) by individuals working on-site yielded incremental lifetime cancer risks ranging from 2E-6 to 5E-5.

Potential exposure to radionuclide COCs (particularly external exposure to radium 226+D) yielded incremental lifetime cancer risk estimates ranging from 1E-3 to 2E-3.

The risk estimates derived from gamma radiation measurements were consistent with those calculated from the activity levels of specific radionuclides.

CURRENT RESIDENTIAL SCENARIO

Using EPA Region 10 default residential exposure assumptions, the incremental cancer risk estimate associated with exposure to metals exceeded background only at the southern residence (8E-6). Risks associated with exposure to radionuclides did not exceed background at any location.

FUTURE RESIDENTIAL SCENARIO

The incremental cancer risk estimate associated with exposure to metals ranged from 4E-6 at the future Southern II residence to 1E-4 at the future Northern I residence. Hazard quotients of 2 and 1 were associated with potential future ingestion of groundwater at the Southern II residence.

External exposure to radium-226+D dominated the total incremental cancer risks associated with potential future industrial exposure to radionuclides; the total risk estimates ranged from below background at the future Southern II residence to 2E-3 at the future Northern I residence.

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Appendix A

Risk-Based Concentrations

Calculation of Risk Based Concentrations (RBCs)

Table A-1 presents calculated RBCs for chemicals in various media via oral, inhalation and external radiation pathways. Non-radionuclide RBCs (industrial and residential) were based on oral ingestion of water and soil, and inhalation of airborne chemicals. Radionuclide RBCs were based on a combined external and ingestion exposure to soil, as well as direct inhalation of airborne materials. The following calculations were used to determine RBCs:

1. Non-radionuclides

   A. noncarcinogenic RBC's $RBC = (HQxRfD)/IF$

   where HQ = Hazard Quotient (set at 0.1)
   RfD = Reference Dose (oral)
   IF = Intake Factor-specific for scenario and pathway (Appendix Tables B-1, B-3)

   B. carcinogenic RBC's $RBC = Risk/(SFxIF)$

   where Risk = carcinogenic risk (set at 10^{-7})
   SF = pathway-specific Slope Factor
   IF = Intake Factor-specific for scenario and pathway (Appendix Tables B-1, B-3)

2. Radionuclides

   A. Soils $RBC = Risk/((SF_O x IF_O)+(SF_E x EF))$

   where Risk = carcinogenic risk (set at 10^{-7})
   SF = pathway-specific Slope Factor
   IF = Intake Factor-specific for scenario and pathway (Appendix Tables B-1, B-3)
   EF = Exposure Factor (Appendix Tables B-1, B-3)

   B. Air $RBC = Risk/(SFxIF)$

   where Risk = carcinogenic risk (set at 10^{-7})
   SF = pathway-specific Slope Factor
   IF = Intake Factor-specific for scenario and pathway (Appendix Tables B-1, B-3)

Table A-1
Risk-Based Concentrations in Water, Soil, and Air for Residential and Industrial Scenarios

	Noncarcinogenic				Carcinogenic				Noncarcinogenic RBCs				Carcinogenic RBCs			
	(mg/kg-day)		(mg/m3)		(mg/kg-day)-1				Water (mg/l) (resident) HQ=0.1	Soil (mg/kg) (resident) HQ=0.1	Soil (mg/kg) (Indust.) HQ=0.1	Air (mg/m3) (resident) HQ=0.1	Water (mg/l) (resident) Risk=1E-7	Soil (mg/kg) (resident) Risk=1E-7	Soil (mg/kg) (Indust.) Risk=1E-7	Air (mg/m3) (resident) Risk=1E-7
ANALYTE	Oral RfD	Source	Inhal. RfC	Source	Oral SF	Source	Inhal. SF	Source								
Aluminum	1.0E+00	3							3.6E+00	2.7E+04	2.0E+05					
Arsenic	3.0E-04	1			1.75[a]		5.0E+01	2	1.1E-03	8.2E+00	6.1E+01		4.9E-06	3.7E-02	3.3E-01	1.7E-08
Beryllium	5.0E-03	1			4.3	1	8.4E+00	2	1.8E-02	1.4E+02	1.0E+03		2.0E-06	1.5E-02	1.3E-01	1.0E-07
Cadmium (water)	5.0E-04	1					6.1E+00	2	1.8E-03	1.4E+01	1.0E+02					1.4E-07
Cadmium (diet)	1.0E-03	1							3.6E-03	2.7E+01	2.0E+02					
Chloride	8.6E+01	4							3.1E+02	2.4E+06	1.8E+07					
Chromium (III)	1.0E+00	1							3.6E+00	2.7E+04	2.0E+05					
Copper	3.7E-02	2							1.4E-01	1.0E+03	7.6E+03					
Fluoride	6.0E-02	1							2.2E-01	1.6E+03	1.2E+04					
Lead	NA	1														
Manganese (diet)	1.4E-01	1	4.0E-04	1						3.8E+03	2.9E+04	9.1E+02				
Manganese (water)	5.0E-03	1							1.8E-02							
Molybdenum	5.0E-03	1							1.8E-02	1.4E+02	1.0E+03					
Nickel, Soluble Salts	2.0E-02	1					8.4E-01	2	7.3E-02	5.5E+02	4.1E+03					1.0E-06
Nitrate as N	1.6E+00	1							5.8E+00	4.4E+04	3.3E+05					
Potassium	--															
Selenium	5.0E-03	1							1.8E-02	1.4E+02	1.0E+03					
Silver	5.0E-03	1							1.8E-02	1.4E+02	1.0E+03					
Sulfate Ion	--															
Uranium	3.2E-03	2								9E+01	6.7E+02					
Vanadium	7.0E-03	2							2.6E-02	1.9E+02	1.4E+03					
Zinc	3.0E-01	1							1.1E+00	8.2E+03	6.1E+04					

Table A-1 (Continued)
Calculation of Risk-Based Concentrations of Radionuclides in Soil and Air
Residential and Industrial Scenarios

Radionuclide	Oral SF (risk/pCi)	Inhal. SF (risk/pCi)	Extern. SF (risk/yr/pCi/g)	Soil RBC (pCi/g) Ingest.+External (Residential) Risk = 1E-07	Soil RBC (pCi/g) Ingest.+External (Industrial) Risk = 1E-07	Air RBC (pCi/m3) Inhalation (Residential) Risk = 1E-07
K-40	1.1E-11	7.6E-12	5.4E-07	7.7E-03	4.0E-02	6.3E-02
Pb-210+D	6.6E-10	4.0E-09	1.6E-10	1.2E-01	4.9E-01	1.2E-04
Po-210	1.5E-10	2.6E-09	2.9E-11	5.3E-01	2.1E+00	1.8E-04
Ra-226+D	1.2E-10	3.0E-09	6.0E-06	6.9E-04	3.6E-03	1.6E-04
Ra-228+D	1.0E-10	6.9E-10	2.9E-06	1.4E-03	7.5E-03	6.9E-04
Rn-222+D	1.7E-12	7.7E-12	5.9E-06	7.1E-04	3.7E-03	6.2E-02
Th-228 + D	5.5E-11	7.8E-08	5.6E-06	7.4E-04	3.9E-03	6.1E-06
Th-230	1.3E-11	2.9E-08	5.4E-11	5.7E+00	2.3E+01	1.6E-05
Th-232	1.2E-11	2.8E-08	2.6E-11	6.4E+00	2.6E+01	1.7E-05
U-234	1.6E-11	2.6E-08	3.0E-11	4.8E+00	2.0E+01	1.8E-05
U-235	1.6E-11	2.5E-08	2.4E-07	1.7E-02	9.0E-02	1.9E-05
U-238+D	2.0E-11	2.4E-08	5.1E-08	8.0E-02	4.2E-01	2.0E-05

(a) The value of 1.75 is derived from a unit risk of 5E-5 $(ug/l)^{-1}$ in drinking water.

(1) IRIS DATABASE, 5/94

(2) HEAST TABLES, 3/92, 3/93, 3/94

(3) USEPA ECAO MEMO

(4) Food and Nutrition Board, National Research Council.

SF = Slope Factor (Values derived from the Health Effects Assessment Summary Tables, 3/94).

RBC = Risk-based concentration for radionuclides in residential media, using revised equation from the RAGS, Part B, p. 35-39, as amended August 1992.

Appendix B

Calculations of Intake Factors, Hazards, and Risks

This appendix calculates hazards and risks for all media/COPC combinations as described in Sections 2, 3 and 5. To facilitate these computations, exposure constants have been consolidated into Intake Factors or Exposure Factors for re-application into hazard and risk equations (Section 3.5). Tables B-1 and B-3 show the calculations of default factors for Industrial and Residential scenarios, respectively. Tables B-2 and B-4 calculate the hazards and risks in industrial and residential scenarios, respectively.

In general, the hazard and risk equations can be summarized as follows:

(1) Noncancer Hazard (metals)= C x IF / RfD

(2) Cancer Risk (Metals) = C x IF x SF

(3) Cancer Risk (Radionuclides) = Oral + External + Inhalation Cancer Risks

(3a) Oral Cancer Risk = $C \times IF_r \times SF_o$
(3b) External Cancer Risk = $C \times EF_r \times SF_e$
(3c) Inhalation Cancer Risk = $C \times IF_i$

where: C = RME or Average (AVG) Concentration
IF = Intake Factor (Tables B-1 and B-3)
EF = Exposure Factor (Tables B-1 and B-3)
RfD = Oral Reference Dose (Appendix A)
SF = Slope Factor (Appendix A)

The Reasonable Maximum Exposure (RME) concentration is determined by taking the lesser value of either the maximum measured concentration (Max.) or the 95% Upper Confidence Limit of the mean concentration (UCL). The UCL is calculated as follows:

$$UCL = avg + (t_{0.5} \times (s/(n-1)\text{^}0.5))$$

The AVG concentration is represented by an arithmetic mean.

The tables are organized as follows:

Industrial	Table B-1a,b,c,d,e,f,g - Industrial Intake/Exposure Factors
	Table B-2a - Future Industrial Risks Calculations
	Table B-2b - Current Industrial Risks Calculations
Residential	Table B-3 Residential Intake/Exposure Factors
	Tables B-4a,b,c, - Current Residential Risk Calculations
	Tables B-4d,e,f,g - Future Residential Risks

Table B-2a calculates default exposure hazards and risks for the various source areas mentioned in Section 3. These results are applied to the future industrial scenario. Table B-2b calculates modified exposure (non-default) hazards and risks, used in the current industrial scenario. A scaling factor was used in Table B-2b for ease of computation. Given equations (1) and (2) above, it can be seen that a chemical specific risk in any given pathway is directly proportional to the intake (or exposure) factor for that pathway. Due to proportionality, any modifications to the intake factor lead to identical scaling of risk; hence scaling factors. In this risk assessment, all pathway factors are scaled equally, within a specific scenario (see Section 3).

For example, at the baghouse dusts area, soil ingestion, dust inhalation and the gamma exposure are all 50% (0.5) of default (Tables 3-2a,b). From the equations in Section 3.5, it can be shown that the corresponding intake and exposure factors are also 50% of default; hence a global scaling factor of 0.5.

At the underflow solids, the only modified exposure is Exposure Frequency (EF). A scaling factor of 0.72 is developed by taking a ratio of the modified parameter (180 d/yr) to the default parameter (250 d/yr). Because EF relates to all exposure pathways (Section 3.5), only one scaling factor per scenario is presented in Table B-2b. All scaling is based on a ratio of modified exposure to default exposure. Other source areas are scaled similarly.

Table B-1a
Calculation of Intake and Exposure Factors for the Current Industrial Scenario - Baghouse Dusts Area

Oral Intake Factors - inorganics in soils

Intake Factor = (Ingestion Rate x Exposure Frequency x Exposure Duration)/(Averaging Time x Body Weight x 10^6 mg/kg)

Hazard or Risk	Ingestion Rate (mg/d)	Exposure Frequency (d/yr)	Exposure Duration (yr)	Averaging Time (d)	Body Weight (kg)	Intake Factor (1/d)
Non-cancer	9	150(a)	25	9,125	70	5.3E-08
Cancer	9	150(a)	25	25,550	70	1.9E-08

Oral Intake Factor - radionuclides in soil

Intake Factor = Ingestion Rate x Exposure Frequency x Exposure Duration x Conversion Factor

Hazard or Risk	Ingestion Rate (mg/d)	Exposure Frequency (d/yr)	Exposure Duration (yr)	Conversion Factor (g/mg)		Intake Factor (1/d)
Cancer	9	150(a)	25	0.001		33.8

External Exposure Factor - radionuclides

Exposure Factor = Exposure Duration x (1-Gamma Shielding Factor) x Gamma Exposure Factor

Hazard or Risk	Exposure Duration (yr)	Gamma Shielding Factor			Gamma Exposure Time factor	Exposure Factor (yr)
Cancer	25	0.0(b)			0.04(c)	1.07

Inhalation Intake Factor - inorganics in air

Intake Factor = (Inhalation Rate x Exposure Frequency x Exposure Duration)/(Averaging Time x Body Weight)

Hazard or Risk	Inhalation Rate (m^3/d)	Exposure Frequency (d/yr)	Exposure Duration (yr)	Averaging Time (d)	Body Weight (kg)	Intake Factor (m^3/kg-d)
Non-cancer	4	250(d)	25	9,125	70	0.04
Cancer	4	250(d)	25	25,550	70	0.01

Inhalation Intake Factors - radionuclides in air

Intake Factor = Exposure Duration x Exposure Frequency x Inhalation Rate

Hazard or Risk	Exposure Duration (yr)	Exposure Frequency (d/yr)	Inhalation Rate (m^3/d)			Intake Factor (m^3)
Cancer	25	250(d)	4			25,000

Shading highlights the calculated values

(a) Workers assumed to be in direct contact with source materials for a fraction of the year because of snow cover.

(b) Workers assumed to be unshielded in the current industrial scenario.

(c) Gamma exposure factor derived: ((1.5 h/d x 250 d/yr x 25 yr)/(24 h/d x 365 d/yr x 25 yr))

(d) Workers assumed to be exposed to radionuclide emissions in source materials and all COCs in air year-round.

Table B-1b
Calculation of Intake and Exposure Factors for the Current Industrial Scenario - Nodules Area

Oral Intake Factors - inorganics in soils

Intake Factor = (Ingestion Rate x Exposure Frequency x Exposure Duration)/(Averaging Time x Body Weight x 10^6 mg/kg)

Hazard or Risk	Ingestion Rate (mg/d)	Exposure Frequency (d/yr)	Exposure Duration (yr)	Averaging Time (d)	Body Weight (kg)	Intake Factor (1/d)
Non-cancer	9	150(a)	25	9,125	70	5.3E-08
Cancer	9	150(a)	25	25,550	70	1.9E-08

Oral Intake Factor - radionuclides in soil

Intake Factor = Ingestion Rate x Exposure Frequency x Exposure Duration x Conversion Factor

Hazard or Risk	Ingestion Rate (mg/d)	Exposure Frequency (d/yr)	Exposure Duration (yr)	Conversion Factor (g/mg)		Intake Factor (1/d)
Cancer	9	150(a)	25	0.001		33.8

External Exposure Factor - radionuclides

Exposure Factor = Exposure Duration x (1-Gamma Shielding Factor) x Gamma Exposure Factor

Hazard or Risk	Exposure Duration (yr)	Gamma Shielding Factor			Gamma Exposure Time factor	Exposure Factor (yr)
Cancer	25	0.0(b)			0.04(c)	1.07

Inhalation Intake Factor - inorganics in air

Intake Factor = (Inhalation Rate x Exposure Frequency x Exposure Duration/(Averaging Time x Body Weight)

Hazard or Risk	Inhalation Rate (m^3/d)	Exposure Frequency (d/yr)	Exposure Duration (yr)	Averaging Time (d)	Body Weight (kg)	Intake Factor (m^3/kg-d)
Non-cancer	4	250(d)	25	9,125	70	0.04
Cancer	4	250(d)	25	25,550	70	0.01

Inhalation Intake Factors - radionuclides in air

Intake Factor = (Exposure Duration x Exposure Frequency x Inhalation Rate)

Hazard or Risk	Exposure Duration (yr)	Exposure Frequency (d/yr)	Inhalation Rate (m^3/d)			Intake Factor (m^3)
Cancer	25	250(d)	4			25,000

Shading highlights the calculated values

(a) Workers assumed to be in direct contact with source materials for a fraction of the year because of snow cover.

(b) Workers assumed to be unshielded in the current industrial scenario.

(c) Gamma exposure factor derived: ((1.5 h/d x 250 d/yr x 25 yr)/(24 h/d x 365 d/yr x 25 yr))

(d) Workers assumed to be exposed to radionuclide emissions in source materials and all COCs in air year-round.

Table B-1c
Calculation of Intake and Exposure Factors for the Current Industrial Scenario - Slag Area

Oral Intake Factors - inorganics in soils

Intake Factor = (Ingestion Rate x Exposure Frequency x Exposure Duration)/(Averaging Time x Body Weight x 10^6 mg/kg)

Hazard or Risk	Ingestion Rate (mg/d)	Exposure Frequency (d/yr)	Exposure Duration (yr)	Averaging Time (d)	Body Weight (kg)	Intake Factor (1/d)
Non-cancer	25	150(a)	25	9,125	70	1.5E-07
Cancer	25	150(a)	25	25,550	70	5.2E-08

Oral Intake Factor - radionuclides in soil

Intake Factor = Ingestion Rate x Exposure Frequency x Exposure Duration x Conversion Factor

Hazard or Risk	Ingestion Rate (mg/d)	Exposure Frequency (d/yr)	Exposure Duration (yr)	Conversion Factor (g/mg)		Intake Factor (1/d)
Cancer	25	150(a)	25	0.001		93.8

External Exposure Factor - radionuclides

Exposure Factor = Exposure Duration x (1-Gamma Shielding Factor) x Gamma Exposure Factor

Hazard or Risk	Exposure Duration (yr)	Gamma Shielding Factor			Gamma Exposure Time factor	Exposure Factor (yr)
Cancer	25	0.45(b)			0.11(c)	1.57

Inhalation Intake Factor - inorganics in air

Intake Factor = (Inhalation Rate x Exposure Frequency x Exposure Duration/(Averaging Time x Body Weight)

Hazard or Risk	Inhalation Rate (m^3/d)	Exposure Frequency (d/yr)	Exposure Duration (yr)	Averaging Time (d)	Body Weight (kg)	Intake Factor (m^3/kg-d)
Non-cancer	5	250(d)	25	9,125	70	0.05
Cancer	5	250(d)	25	25,550	70	0.02

Inhalation Intake Factors - radionuclides in air

Intake Factor = (Exposure Duration x Exposure Frequency x Inhalation Rate)

Hazard or Risk	Exposure Duration (yr)	Exposure Frequency (d/yr)	Inhalation Rate (m^3/d)			Intake Factor (m^3)
Cancer	25	250(d)	5			31,250

Shading highlights the calculated values

(a) Workers assumed to be in direct contact with source materials for a fraction of the year because of snow cover.

(b) Workers assumed to be shielded in the current industrial scenario.

(c) Gamma exposure factor derived: ((4 h/d x 250 d/yr x 25 yr)/(24 h/d x 365 d/yr x 25 yr))

(d) Workers assumed to be exposed to radionuclide emissions in source materials and all COCs in air year-round.

Table B-1d
Calculation of Intake and Exposure Factors for the Current Industrial Scenario - Road Dusts Area

Oral Intake Factors - inorganics in soils

Intake Factor = (Ingestion Rate x Exposure Frequency x Exposure Duration)/(Averaging Time x Body Weight x 10^6 mg/kg)

Hazard or Risk	Ingestion Rate (mg/d)	Exposure Frequency (d/yr)	Exposure Duration (yr)	Averaging Time (d)	Body Weight (kg)	Intake Factor (1/d)
Non-cancer	37.5	96(a)	25	9,125	70	1.4E-07
Cancer	37.5	96(a)	25	25,550	70	5.0E-08

Oral Intake Factor - radionuclides in soil

Intake Factor = Ingestion Rate x Exposure Frequency x Exposure Duration x Conversion Factor

Hazard or Risk	Ingestion Rate (mg/d)	Exposure Frequency (d/yr)	Exposure Duration (yr)	Conversion Factor (g/mg)		Intake Factor (1/d)
Cancer	37.5	96(a)	25	0.001		90.0

External Exposure Factor - radionuclides

Exposure Factor = Exposure Duration x (1-Gamma Shielding Factor) x Gamma Exposure Factor

Hazard or Risk	Exposure Duration (yr)	Gamma Shielding Factor			Gamma Exposure Time factor	Exposure Factor (yr)
Cancer	25	0.45(b)			0.11(c)	1.51

Inhalation Intake Factor - inorganics in air

Intake Factor = (Inhalation Rate x Exposure Frequency x Exposure Duration/(Averaging Time x Body Weight)

Hazard or Risk	Inhalation Rate (m^3/d)	Exposure Frequency (d/yr)	Exposure Duration (yr)	Averaging Time (d)	Body Weight (kg)	Intake Factor (m^3/kg-d)
Non-cancer	15	160(d)	25	9,125	70	0.09
Cancer	15	160(d)	25	25,550	70	0.03

Inhalation Intake Factors - radionuclides in air

Intake Factor = (Exposure Duration x Exposure Frequency x Inhalation Rate)

Hazard or Risk	Exposure Duration (yr)	Exposure Frequency (d/yr)	Inhalation Rate (m^3/d)			Intake Factor (m^3)
Cancer	25	160(d)	15			60,000

Shading highlights the calculated values

(a) Workers assumed to be in direct contact with source materials for a fraction of the year because of snow cover.

(b) Workers assumed to be shielded in the current industrial scenario.

(c) Gamma exposure factor derived: ((6 h/d x 160 d/yr x 25 yr)/(24 h/d x 365 d/yr x 25 yr))

(d) Workers assumed to be exposed to radionuclide emissions in source materials and all COCs in air year-round.

Table B-1e
Calculation of Intake and Exposure Factors for the Current Industrial Scenario - Treater Dusts Area

Oral Intake Factors - inorganics in soils

Intake Factor = (Ingestion Rate x Exposure Frequency x Exposure Duration)/(Averaging Time x Body Weight x 10^6 mg/kg)

Hazard or Risk	Ingestion Rate (mg/d)	Exposure Frequency (d/yr)	Exposure Duration (yr)	Averaging Time (d)	Body Weight (kg)	Intake Factor (1/d)
Non-cancer	6.25	150(a)	25	9,125	70	3.7E-08
Cancer	6.25	150(a)	25	25,550	70	1.3E-08

Oral Intake Factor - radionuclides in soil

Intake Factor = Ingestion Rate x Exposure Frequency x Exposure Duration x Conversion Factor

Hazard or Risk	Ingestion Rate (mg/d)	Exposure Frequency (d/yr)	Exposure Duration (yr)	Conversion Factor (g/mg)		Intake Factor (1/d)
Cancer	6.25	150(a)	25	0.001		23.4

External Exposure Factor - radionuclides

Exposure Factor = Exposure Duration x (1-Gamma Shielding Factor) x Gamma Exposure Factor

Hazard or Risk	Exposure Duration (yr)	Gamma Shielding Factor			Gamma Exposure Time factor	Exposure Factor (yr)
Cancer	25	0.45(b)			0.03(c)	0.39

Inhalation Intake Factor - inorganics in air

Intake Factor = (Inhalation Rate x Exposure Frequency x Exposure Duration/(Averaging Time x Body Weight)

Hazard or Risk	Inhalation Rate (m^3/d)	Exposure Frequency (d/yr)	Exposure Duration (yr)	Averaging Time (d)	Body Weight (kg)	Intake Factor (m^3/kg-d)
Non-cancer	2.5	250(d)	25	9,125	70	0.02
Cancer	2.5	250(d)	25	25,550	70	0.01

Inhalation Intake Factors - radionuclides in air

Intake Factor = (Exposure Duration x Exposure Frequency x Inhalation Rate)

Hazard or Risk	Exposure Duration (yr)	Exposure Frequency (d/yr)	Inhalation Rate (m^3/d)			Intake Factor (m^3)
Cancer	25	250(d)	2.5			15,625

Shading highlights the calculated values

(a) Workers assumed to be in direct contact with source materials for a fraction of the year because of snow cover.

(b) Workers assumed to be shielded in the current industrial scenario.

(c) Gamma exposure factor derived: ((1 h/d x 250 d/yr x 25 yr)/(24 h/d x 365 d/yr x 25 yr))

(d) Workers assumed to be exposed to radionuclide emissions in source materials and all COCs in air year-round.

Table B-1f
Calculation of Intake and Exposure Factors for the Current Industrial Scenario - Underflow Solids Area

Oral Intake Factors - inorganics in soils

Intake Factor = (Ingestion Rate x Exposure Frequency x Exposure Duration)/(Averaging Time x Body Weight x 10^6 mg/kg)

Hazard or Risk	Ingestion Rate (mg/d)	Exposure Frequency (d/yr)	Exposure Duration (yr)	Averaging Time (d)	Body Weight (kg)	Intake Factor (1/d)
Non-cancer	37.5	108(a)	25	9,125	70	1.6E-07
Cancer	37.5	108(a)	25	25,550	70	5.7E-08

Oral Intake Factor - radionuclides in soil

Intake Factor = Ingestion Rate x Exposure Frequency x Exposure Duration x Conversion Factor

Hazard or Risk	Ingestion Rate (mg/d)	Exposure Frequency (d/yr)	Exposure Duration (yr)	Conversion Factor (g/mg)		Intake Factor (1/d)
Cancer	37.5	108(a)	25	0.001		101.3

External Exposure Factor - radionuclides

Exposure Factor = Exposure Duration x (1-Gamma Shielding Factor) x Gamma Exposure Factor

Hazard or Risk	Exposure Duration (yr)	Gamma Shielding Factor			Gamma Exposure Time factor	Exposure Factor (yr)
Cancer	25	0.45(b)			0.12(c)	1.70

Inhalation Intake Factor - inorganics in air

Intake Factor = (Inhalation Rate x Exposure Frequency x Exposure Duration/(Averaging Time x Body Weight)

Hazard or Risk	Inhalation Rate (m^3/d)	Exposure Frequency (d/yr)	Exposure Duration (yr)	Averaging Time (d)	Body Weight (kg)	Intake Factor (m^3/kg-d)
Non-cancer	15	180(d)	25	9,125	70	0.11
Cancer	15	180(d)	25	25,550	70	0.04

Inhalation Intake Factors - radionuclides in air

Intake Factor = (Exposure Duration x Exposure Frequency x Inhalation Rate)

Hazard or Risk	Exposure Duration (yr)	Exposure Frequency (d/yr)	Inhalation Rate (m^3/d)			Intake Factor (m^3)
Cancer	25	180(d)	15			67,500

Shading highlights the calculated values

(a) Workers assumed to be in direct contact with source materials for a fraction of the year because of snow cover.

(b) Workers assumed to be shielded in the current industrial scenario.

(c) Gamma exposure factor derived: ((6 h/d x 180 d/yr x 25 yr)/(24 h/d x 365 d/yr x 25 yr))

(d) Workers assumed to be exposed to radionuclide emissions in source materials and all COCs in air year-round.

Table B-1g
Calculation of Intake and Exposure Factors for the Future Industrial Scenario

Calculation of Oral Intake Factor (inorganics in soils)

Intake Factor = (Ingestion Rate x Exposure Frequency x Exposure Duration)/(Averaging Time x Body Weight x 10^6 mg/kg)

Hazard or Risk	Ingestion Rate (mg/d)	Exposure Frequency (d/yr)	Exposure Duration (yr)	Averaging Time (d)	Body Weight (kg)	RME Intake Factor (1/d)
Non-cancer	50	150(a)	25	9,125	70	2.9E-07
Cancer	50	150(a)	25	25,550	70	1.0E-07

Calculation of Oral Intake Factor (radionuclides in soil)

Intake Factor = Ingestion Rate x Exposure Frequency x Exposure Duration x Conversion Factor

Hazard or Risk	Ingestion Rate (mg/d)	Exposure Frequency (d/yr)	Exposure Duration (yr)	Conversion Factor (g/mg)		RME Intake Factor (1/d)
Cancer	50	150(a)	25	0.001		187.5

Calculation of External Exposure Factor (radionuclides)

Exposure Factor = Exposure Duration x (1-Gamma Shielding Factor) x Gamma Exposure Factor

Hazard or Risk	Exposure Duration (yr)	Gamma Shielding Factor			Gamma Exposure Time factor	Exposure Factor (yr)
Cancer	25	0(b)			0.24(c)	6.00

Calculation of Inhalation Intake Factor (inorganics in air)

Intake Factor = (Inhalation Rate x Exposure Frequency x Exposure Duration)/(Averaging Time x Body Weight)

Hazard or Risk	Inhalation Rate (m^3/d)	Exposure Frequency (d/yr)	Exposure Duration (yr)	Averaging Time (d)	Body Weight (kg)	RME Intake Factor (m^3/kg-d)
Non-cancer	20	250(d)	25	9,125	70	0.20
Cancer	20	250(d)	25	25,550	70	0.07

Calculation of Inhalation Intake Factors (radionuclides in air)

Intake Factor = Exposure Duration x Exposure Frequency x Inhalation Rate

Hazard or Risk	Exposure Duration (yr)	Exposure Frequency (d/yr)	Inhalation Rate (m^3/d)			RME Intake Factor (m^3)
Cancer	25	250(d)	20			125,000

Shading highlights the calculated values

(a) Workers assumed to be in direct contact with source materials for a fraction of the year because of snow cover.

(b) Workers assumed to be unshielded in the future industrial scenario.

(c) Gamma factor derived: ((8 h/d x 250 d/yr x 25 yr)/(24 h/d x 365 d/yr x 25 yr))

(d) Workers assumed to be exposed to radionuclide emissions in source materials or COCs in air year-round.

Table B-2a
Calculation[a] of Hazards and Risks at Onsite Source Areas
Future Industrial Scenario

	Concentration		Factors (Table B-1)					Reference Doses (RfD) and Slope Factors (SF)				Hazard Quotients and Cancer Risks				
			Oral Intake			External	Inhalation	Non-Cancer	Cancer - Metals/Rads			Hazard	Cancer Risk			
			Metals		Radionuclides	Exposure	Intake	RfDs	SFs							
COC	Source	Air	Non-Cancer	Cancer	Cancer	Cancer	Cancer	Oral RfD	Oral	External	Inhalation	Quotient	Ingestion	External	Inhalation	TOTAL
BAGHOUSE DUST AREA																
Arsenic	85	2E-07	2.9E-07	1.0E-07	--	--	7.0E-02	3E-04	2E+00	--	5E+01	8E-02	2E-05	--	6E-07	2E-05
Beryllium	4.7	8E-08	2.9E-07	1.0E-07	--	--	7.0E-02	5E-03	4E+00	--	8E+00	3E-04	2E-06	--	5E-08	2E-06
Cadmium	504	7E-08	2.9E-07	1.0E-07	--	--	7.0E-02	1E-03	--	--	6E+00	1E-01	--	--	3E-08	3E-08
Vanadium	769	2E-05	2.9E-07	1.0E-07	--	--	7.0E-02	7E-03	--	--	--	3E-02	--	--	--	--
												Pathway Risk	2E-05	--	7E-07	2E-05
RADIONUCLIDES																
Lead-210+D	100	2E-03	--	--	1.9E+02	6.0E+00	1.3E+05	--	7E-10	2E-10	4E-09	--	1E-05	1E-07	8E-07	1E-05
Radium-226+D	32	3E-04	--	--	1.9E+02	6.0E+00	1.3E+05	--	1E-10	6E-06	3E-09	--	7E-07	1E-03	1E-07	1E-03
Thorium-230	33	3E-04	--	--	1.9E+02	6.0E+00	1.3E+05	--	1E-11	5E-11	3E-08	--	8E-08	1E-08	1E-06	1E-06
Uranium-238+D	35	3E-04	--	--	1.9E+02	6.0E+00	1.3E+05	--	2E-11	5E-08	2E-08	--	1E-07	1E-05	1E-06	1E-05
												Pathway Risk	1E-05	1E-03	3E-06	1E-03

(a) Units are not shown for concentrations, factors, or toxicity values; units are listed in Table B-3, Appendix A, Section 2.0.

Table B-2a (Continued)
Calculation[a] of Hazards and Risks at Onsite Source Areas
Future Industrial Scenario

	Concentration		Factors (Table B-1)					Reference Doses (RfD) and Slope Factors (SF)				Hazard Quotients and Cancer Risks				
			Oral Intake			External	Inhalation	Non-Cancer	Cancer - Metals/Rads							
			Metals		Radionuclides	Exposure	Intake	RfDs	SFs			Hazard	Cancer Risk			
COC	Source	Air	Non-Cancer	Cancer	Cancer	Cancer	Cancer	Oral RfD	Oral	External	Inhalation	Quotient	Ingestion	External	Inhalation	TOTAL
NODULES																
Arsenic	11	2E-07	2.9E-07	1.0E-07	--	--	7.0E-02	3E-04	2E+00	--	5E+01	1E-02	2E-06	--	6E-07	3E-06
Beryllium	14	8E-08	2.9E-07	1.0E-07	--	--	7.0E-02	5E-03	4E+00	--	8E+00	8E-04	6E-06	--	5E-08	6E-06
Cadmium	BB	7E-08	2.9E-07	1.0E-07	--	--	7.0E-02	1E-03	--	--	6E+00	--	--	--	3E-08	3E-08
Vanadium	1640	2E-05	2.9E-07	1.0E-07	--	--	7.0E-02	7E-03	--	--	--	7E-02	--	--	--	--
												Pathway Risk	8E-06	--	7E-07	9E-06
RADIONUCLIDES																
Lead-210+D	BB	2E-03	--	--	1.9E+02	6.0E+00	1.3E+05	--	7E-10	2E-10	4E-09	--	--	--	8E-07	8E-07
Radium-226+D	51	3E-04	--	--	1.9E+02	6.0E+00	1.3E+05	--	1E-10	6E-06	3E-09	--	1E-06	2E-03	1E-07	2E-03
Thorium-230	54	3E-04	--	--	1.9E+02	6.0E+00	1.3E+05	--	1E-11	5E-11	3E-08	--	1E-07	2E-08	1E-06	1E-06
Uranium-238+D	48	3E-04	--	--	1.9E+02	6.0E+00	1.3E+05	--	2E-11	5E-08	2E-08	--	2E-07	1E-05	1E-06	2E-05
												Pathway Risk	1E-06	2E-03	3E-06	2E-03

(a) Units are not shown for concentrations, factors, or toxicity values; units are listed in Table B-3, Appendix A, Section 2.0.

BB = Below background

Table B-2a (Continued)
Calculation[a] of Hazards and Risks at Onsite Source Areas
Future Industrial Scenario

			Factors (Table B-1)					Reference Doses (RfD) and Slope Factors (SF)				Hazard Quotients and Cancer Risks				
	Concentration		Oral Intake			External	Inhalation	Non-Cancer	Cancer - Metals/Rads			Hazard	Cancer Risk			
			Metals		Radionuclides	Exposure	Intake	RfDs	SFs							
COC	Source	Air	Non-Cancer	Cancer	Cancer	Cancer	Cancer	Oral RfD	Oral	External	Inhalation	Quotient	Ingestion	External	Inhalation	TOTAL
SLAG																
Arsenic	6.4	2E-07	2.9E-07	1.0E-07	--	--	7.0E-02	3E-04	2E+00	--	5E+01	6E-03	1E-06	--	6E-07	2E-06
Beryllium	9.9	8E-08	2.9E-07	1.0E-07	--	--	7.0E-02	5E-03	4E+00	--	8E+00	6E-04	4E-06	--	5E-08	5E-06
Cadmium	35	7E-08	2.9E-07	1.0E-07	--	--	7.0E-02	1E-03	--	--	6E+00	1E-02	--	--	3E-08	3E-08
Vanadium	524	2E-05	2.9E-07	1.0E-07	--	--	7.0E-02	7E-03	--	--	--	2E-02	--	--	--	--
												Pathway Risk	6E-06	--	7E-07	6E-06
RADIONUCLIDES																
Lead-210+D	6.6	2E-03	--	--	1.9E+02	6.0E+00	1.3E+05	--	7E-10	2E-10	4E-09	--	8E-07	6E-09	8E-07	2E-06
Radium-226+D	50	3E-04	--	--	1.9E+02	6.0E+00	1.3E+05	--	1E-10	6E-06	3E-09	--	1E-06	2E-03	1E-07	2E-03
Thorium-230	50	3E-04	--	--	1.9E+02	6.0E+00	1.3E+05	--	1E-11	5E-11	3E-08	--	1E-07	2E-08	1E-06	1E-06
Uranium-238+D	45	3E-04	--	--	1.9E+02	6.0E+00	1.3E+05	--	2E-11	5E-08	2E-08	--	2E-07	1E-05	1E-06	1E-05
												Pathway Risk	2E-06	2E-03	3E-06	2E-03

(a) Units are not shown for concentrations, factors, or toxicity values; units are listed in Table B-3, Appendix A, Section 2.0.

Table B-2a (Continued)
Calculation[a] of Hazards and Risks at Onsite Source Areas
Future Industrial Scenario

			Factors (Table B-1)					Reference Doses (RfD) and Slope Factors (SF)				Hazard Quotients and Cancer Risks				
			Oral Intake			External	Inhalation	Non-Cancer	Cancer - Metals/Rads							
	Concentration		Metals		Radionuclides	Exposure	Intake	RfDs	SFs			Hazard	Cancer Risk			
COC	Source	Air	Non-Cancer	Cancer	Cancer	Cancer	Cancer	Oral RfD	Oral	External	Inhalation	Quotient	Ingestion	External	Inhalation	TOTAL
ROAD DUSTS																
Arsenic	43	2E-07	2.9E-07	1.0E-07	--	--	7.0E-02	3E-04	2E+00	--	5E+01	4E-02	8E-06	--	6E-07	8E-06
Beryllium	3.6	8E-08	2.9E-07	1.0E-07	--	--	7.0E-02	5E-03	4E+00	--	8E+00	2E-04	2E-06	--	5E-08	2E-06
Cadmium	483	7E-08	2.9E-07	1.0E-07	--	--	7.0E-02	1E-03	--	--	6E+00	1E-01	--	--	3E-08	3E-08
Vanadium	1010	2E-05	2.9E-07	1.0E-07	--	--	7.0E-02	7E-03	--	--	--	4E-02	--	--	--	--
												Pathway Risk	1E-05	--	7E-07	1E-05
RADIONUCLIDES																
Lead-210+D	190	2E-03	--	--	1.9E+02	6.0E+00	1.3E+05	--	7E-10	2E-10	4E-09	--	2E-05	2E-07	8E-07	2E-05
Radium-226+D	42	3E-04	--	--	1.9E+02	6.0E+00	1.3E+05	--	1E-10	6E-06	3E-09	--	9E-07	2E-03	1E-07	2E-03
Thorium-230	44	3E-04	--	--	1.9E+02	6.0E+00	1.3E+05	--	1E-11	5E-11	3E-08	--	1E-07	1E-08	1E-06	1E-06
Uranium-238+D	43	3E-04	--	--	1.9E+02	6.0E+00	1.3E+05	--	2E-11	5E-08	2E-08	--	2E-07	1E-05	1E-06	1E-05
												Pathway Risk	2E-05	2E-03	3E-06	2E-03

(a) Units are not shown for concentrations, factors, or toxicity values; units are listed in Table B-3, Appendix A, Section 2.0.

Table B-2a (Continued)
Calculation[a] of Hazards and Risks at Onsite Source Areas
Future Industrial Scenario

			Factors (Table B-1)					Reference Doses (RfD) and Slope Factors (SF)				Hazard Quotients and Cancer Risks				
			Oral Intake			External	Inhalation	Non-Cancer	Cancer - Metals/Rads							
	Concentration		Metals		Radionuclides	Exposure	Intake	RfDs	SFs			Hazard	Cancer Risk			
COC	Source	Air	Non-Cancer	Cancer	Cancer	Cancer	Cancer	Oral RfD	Oral	External	Inhalation	Quotient	Ingestion	External	Inhalation	TOTAL
TREATER DUST AREA																
Arsenic	BB	2E-07	2.9E-07	1.0E-07	--	--	7.0E-02	3E-04	2E+00	--	5E+01	--	--	--	6E-07	6E-07
Beryllium	5.9	8E-08	2.9E-07	1.0E-07	--	--	7.0E-02	5E-03	4E+00	--	8E+00	3E-04	3E-06	--	5E-08	3E-06
Cadmium	134	7E-08	2.9E-07	1.0E-07	--	--	7.0E-02	1E-03	--	--	6E+00	4E-02	--	--	3E-08	3E-08
Vanadium	845	2E-05	2.9E-07	1.0E-07	--	--	7.0E-02	7E-03	--	--		4E-02	--	--	--	--
Fluoride	10628	--	2.9E-07	--	--	--	--	6E-02	--	--	--	5E-02	--	--	--	--
												Pathway Risk	3E-06	--	7E-07	3E-06
RADIONUCLIDES																
Lead-210+D	357	2E-03	--	--	1.9E+02	6.0E+00	1.3E+05	--	7E-10	2E-10	4E-09	--	4E-05	3E-07	8E-07	5E-05
Radium-226+D	27	3E-04	--	--	1.9E+02	6.0E+00	1.3E+05	--	1E-10	6E-06	3E-09	--	6E-07	1E-03	1E-07	1E-03
Thorium-230	27	3E-04	--	--	1.9E+02	6.0E+00	1.3E+05	--	1E-11	5E-11	3E-08	--	7E-08	9E-09	1E-06	1E-06
Uranium-238+D	24	3E-04	--	--	1.9E+02	6.0E+00	1.3E+05	--	2E-11	5E-08	2E-08	--	9E-08	7E-06	1E-06	8E-06
												Pathway Risk	4E-05	1E-03	3E-06	1E-03

(a) Units are not shown for concentrations, factors, or toxicity values; units are listed in Table B-3, Appendix A, Section 2.0.

BB = Below background

Table B-2a (Continued)
Calculation[a] of Hazards and Risks at Onsite Source Areas
Future Industrial Scenario

	Concentration		Factors (Table B-1)					Reference Doses (RfD) and Slope Factors (SF)				Hazard Quotients and Cancer Risks				
			Oral Intake			External	Inhalation	Non-Cancer	Cancer - Metals/Rads				Cancer Risk			
			Metals		Radionuclides	Exposure	Intake	RfDs	SFs			Hazard				
COC	Source	Air	Non-Cancer	Cancer	Cancer	Cancer	Cancer	Oral RfD	Oral	External	Inhalation	Quotient	Ingestion	External	Inhalation	TOTAL
UNDERFLOW SOLIDS AREA																
Arsenic	245	2E-07	2.9E-07	1.0E-07	--	--	7.0E-02	3E-04	2E+00	--	5E+01	2E-01	4E-05	--	6E-07	5E-05
Beryllium	15	8E-08	2.9E-07	1.0E-07	--	--	7.0E-02	5E-03	4E+00	--	8E+00	9E-04	7E-06	--	5E-08	7E-06
Cadmium	1881	7E-08	2.9E-07	1.0E-07	--	--	7.0E-02	1E-03	--	--	6E+00	6E-01	--	--	3E-08	3E-08
Vanadium	2609	2E-05	2.9E-07	1.0E-07	--	--	7.0E-02	7E-03	--	--	--	1E-01	--	--	--	--
												Pathway Risk	5E-05	--	7E-07	5E-05
RADIONUCLIDES																
Lead-210+D	341	2E-03	--	--	1.9E+02	6.0E+00	1.3E+05	--	7E-10	2E-10	4E-09	--	4E-05	3E-07	8E-07	4E-05
Radium-226+D	41	3E-04	--	--	1.9E+02	6.0E+00	1.3E+05	--	1E-10	6E-06	3E-09	--	9E-07	1E-03	1E-07	1E-03
Thorium-230	45	3E-04	--	--	1.9E+02	6.0E+00	1.3E+05	--	1E-11	5E-11	3E-08	--	1E-07	1E-08	1E-06	1E-06
Uranium-238+D	43	3E-04	--	--	1.9E+02	6.0E+00	1.3E+05	--	2E-11	5E-08	2E-08	--	2E-07	1E-05	1E-06	1E-05
												Pathway Risk	4E-05	1E-03	3E-06	2E-03

(a) Units are not shown for concentrations, factors, or toxicity values; units are listed in Table B-3, Appendix A, Section 2.0.

NOTE: The hazard quotient for arsenic, cadmium, and arsenic, if summed, would be greater than 1.0.

Table B-2b
Calculation[a] of Hazards and Risks at Onsite Source Areas
Current Industrial Scenario

	Concentration		Factors (Table B-1)					Reference Doses (RfD) and Slope Factors (SF)				Hazard Quotients and Cancer Risks				
			Oral Intake			External	Inhalation	Non-Cancer	Cancer - Metals/Rads			Hazard	Cancer Risk			
			Metals		Radionuclides	Exposure	Intake	RfDs	SFs							
COC	Source	Air	Non-Cancer	Cancer	Cancer	Cancer	Cancer	Oral RfD	Oral	External	Inhalation	Quotient	Ingestion	External	Inhalation	TOTAL
BAGHOUSE DUST AREA																
Arsenic	85	1E-06	5.3E-08	1.9E-08	--	--	1.4E-02	3E-04	2E+00	--	5E+01	1E-02	3E-06	--	7E-07	3E-06
Beryllium	4.7	1E-07	5.3E-08	1.9E-08	--	--	1.4E-02	5E-03	4E+00	--	8E+00	5E-05	4E-07	--	2E-08	4E-07
Cadmium	504	1E-05	5.3E-08	1.9E-08	--	--	1.4E-02	1E-03	--	--	6E+00	3E-02	--	--	8E-07	8E-07
Vanadium	769	4E-05	5.3E-08	1.9E-08	--	--	1.4E-02	7E-03	--	--	--	6E-03	--	--	--	--
												Pathway Risk	3E-06	--	2E-06	5E-06
RADIONUCLIDES																
Lead-210+D	100	2E-03	--	--	3.4E+01	1.1E+00	2.5E+04	--	7E-10	2E-10	4E-09	--	2E-06	2E-08	2E-07	2E-06
Radium-226+D	32	1E-03	--	--	3.4E+01	1.1E+00	2.5E+04	--	1E-10	6E-06	3E-09	--	1E-07	2E-04	8E-08	2E-04
Thorium-230	33	1E-03	--	--	3.4E+01	1.1E+00	2.5E+04	--	1E-11	5E-11	3E-08	--	1E-08	2E-09	8E-07	8E-07
Uranium-238+D	35	1E-03	--	--	3.4E+01	1.1E+00	2.5E+04	--	2E-11	5E-08	2E-08	--	2E-08	2E-06	6E-07	3E-06
												Pathway Risk	2E-06	2E-04	2E-06	2E-04

(a) Units are not shown for concentrations, factors, or toxicity values; units are listed in Table B-3, Appendix A, Section 2.0.

Table B-2b (Continued)
Calculation[a] of Hazards and Risks at Onsite Source Areas
Current Industrial Scenario

	Concentration		Factors (Table B-1)					Reference Doses (RfD) and Slope Factors (SF)				Hazard Quotients and Cancer Risks				
			Oral Intake			External	Inhalation	Non-Cancer	Cancer - Metals/Rads							
			Metals		Radionuclides	Exposure	Intake	RfDs	SFs			Hazard	Cancer Risk			
COC	Source	Air	Non-Cancer	Cancer	Cancer	Cancer	Cancer	Oral RfD	Oral	External	Inhalation	Quotient	Ingestion	External	Inhalation	TOTAL
NODULES																
Arsenic	11	1E-06	5.3E-08	1.9E-08	--	--	1.4E-02	3E-04	2E+00	--	5E+01	2E-03	4E-07	--	7E-07	1E-06
Beryllium	14	1E-07	5.3E-08	1.9E-08	--	--	1.4E-02	5E-03	4E+00	--	8E+00	1E-04	1E-06	--	2E-08	1E-06
Cadmium	BB	1E-05	5.3E-08	1.9E-08	--	--	1.4E-02	1E-03	--	--	6E+00	--	--	--	8E-07	8E-07
Vanadium	1640	4E-05	5.3E-08	1.9E-08	--	--	1.4E-02	7E-03	--	--	--	1E-02	--	--	--	--
												Pathway Risk	1E-06	--	2E-06	3E-06
RADIONUCLIDES																
Lead-210+D	BB	2E-03	--	--	3.4E+01	1.1E+00	2.5E+04	--	7E-10	2E-10	4E-09	--	--	--	2E-07	2E-07
Radium-226+D	51	1E-03	--	--	3.4E+01	1.1E+00	2.5E+04	--	1E-10	6E-06	3E-09	--	2E-07	3E-04	8E-08	3E-04
Thorium-230	54	1E-03	--	--	3.4E+01	1.1E+00	2.5E+04	--	1E-11	5E-11	3E-08	--	2E-08	3E-09	8E-07	8E-07
Uranium-238+D	48	1E-03	--	--	3.4E+01	1.1E+00	2.5E+04	--	2E-11	5E-08	2E-08	--	3E-08	3E-06	6E-07	3E-06
												Pathway Risk	3E-07	3E-04	2E-06	3E-04

(a) Units are not shown for concentrations, factors, or toxicity values; units are listed in Table B-3, Appendix A, Section 2.0.

BB = Below background

Table B-2b (Continued)
Calculation[a] of Hazards and Risks at Onsite Source Areas
Current Industrial Scenario

	Concentration		Factors (Table B-1)					Reference Doses (RfD) and Slope Factors (SF)				Hazard Quotients and Cancer Risks				
			Oral Intake			External	Inhalation	Non-Cancer	Cancer - Metals/Rads			Hazard	Cancer Risk			
			Metals		Radionuclides	Exposure	Intake	RfDs	SFs							
COC	Source	Air	Non-Cancer	Cancer	Cancer	Cancer	Cancer	Oral RfD	Oral	External	Inhalation	Quotient	Ingestion	External	Inhalation	TOTAL
SLAG																
Arsenic	6.4	1E-06	1.5E-07	5.2E-08	--	--	1.7E-02	3E-04	2E+00	--	5E+01	3E-03	6E-07	--	8E-07	1E-06
Beryllium	9.9	1E-07	1.5E-07	5.2E-08	--	--	1.7E-02	5E-03	4E+00	--	8E+00	3E-04	2E-06	--	2E-08	2E-06
Cadmium	35	1E-05	1.5E-07	5.2E-08	--	--	1.7E-02	1E-03	--	--	6E+00	5E-03	--	--	1E-06	1E-06
Vanadium	524	4E-05	1.5E-07	5.2E-08	--	--	1.7E-02	7E-03	--	--	--	1E-02	--	--	--	--
												Pathway Risk	3E-06	--	2E-06	5E-06
RADIONUCLIDES																
Lead-210+D	6.6	2E-03	--	--	9.4E+01	1.6E+00	3.1E+04	--	7E-10	2E-10	4E-09	--	4E-07	2E-09	3E-07	7E-07
Radium-226+D	50	1E-03	--	--	9.4E+01	1.6E+00	3.1E+04	--	1E-10	6E-06	3E-09	--	6E-07	5E-04	9E-08	5E-04
Thorium-230	50	1E-03	--	--	9.4E+01	1.6E+00	3.1E+04	--	1E-11	5E-11	3E-08	--	6E-08	4E-09	1E-06	1E-06
Uranium-238+D	45	1E-03	--	--	9.4E+01	1.6E+00	3.1E+04	--	2E-11	5E-08	2E-08	--	8E-08	4E-06	7E-07	4E-06
												Pathway Risk	1E-06	5E-04	2E-06	5E-04

(a) Units are not shown for concentrations, factors, or toxicity values; units are listed in Table B-3, Appendix A, Section 2.0.

Table B-2b (Continued)
Calculation[a] of Hazards and Risks at Onsite Source Areas
Current Industrial Scenario

			Factors (Table B-1)					Reference Doses (RfD) and Slope Factors (SF)				Hazard Quotients and Cancer Risks				
			Oral Intake			External	Inhalation	Non-Cancer	Cancer - Metals/Rads							
	Concentration		Metals		Radionuclides	Exposure	Intake	RfDs	SFs			Hazard	Cancer Risk			
COC	Source	Air	Non-Cancer	Cancer	Cancer	Cancer	Cancer	Oral RfD	Oral	External	Inhalation	Quotient	Ingestion	External	Inhalation	TOTAL
ROAD DUSTS																
Arsenic	43	1E-06	1.4E-07	5.0E-08	--	--	3.4E-02	3E-04	2E+00	--	5E+01	2E-02	4E-06	--	2E-06	5E-06
Beryllium	3.6	1E-07	1.4E-07	5.0E-08	--	--	3.4E-02	5E-03	4E+00	--	8E+00	1E-04	8E-07	--	4E-08	8E-07
Cadmium	483	1E-05	1.4E-07	5.0E-08	--	--	3.4E-02	1E-03	--	--	6E+00	7E-02	--	--	2E-06	2E-06
Vanadium	1010	4E-05	1.4E-07	5.0E-08	--	--	3.4E-02	7E-03	--	--	--	2E-02	--	--	--	--
												Pathway Risk	5E-06	--	4E-06	8E-06
RADIONUCLIDES																
Lead-210+D	190	2E-03	--	--	9.0E+01	1.5E+00	6.0E+04	--	7E-10	2E-10	4E-09	--	1E-05	5E-08	6E-07	1E-05
Radium-226+D	42	1E-03	--	--	9.0E+01	1.5E+00	6.0E+04	--	1E-10	6E-06	3E-09	--	5E-07	4E-04	2E-07	4E-04
Thorium-230	44	1E-03	--	--	9.0E+01	1.5E+00	6.0E+04	--	1E-11	5E-11	3E-08	--	5E-08	4E-09	2E-06	2E-06
Uranium-238+D	43	1E-03	--	--	9.0E+01	1.5E+00	6.0E+04	--	2E-11	5E-08	2E-08	--	8E-08	3E-06	1E-06	5E-06
												Pathway Risk	1E-05	4E-04	4E-06	4E-04

(a) Units are not shown for concentrations, factors, or toxicity values; units are listed in Table B-3, Appendix A, Section 2.0.

Table B-2b (Continued)
Calculation[a] of Hazards and Risks at Onsite Source Areas
Current Industrial Scenario

	Concentration		Factors (Table B-1)					Reference Doses (RfD) and Slope Factors (SF)				Hazard Quotients and Cancer Risks				
			Oral Intake			External	Inhalation	Non-Cancer	Cancer - Metals/Rads							
			Metals		Radionuclides	Exposure	Intake	RfDs	SFs			Hazard	Cancer Risk			
COC	Source	Air	Non-Cancer	Cancer	Cancer	Cancer	Cancer	Oral RfD	Oral	External	Inhalation	Quotient	Ingestion	External	Inhalation	TOTAL
TREATER DUST AREA																
Arsenic	BB	1E-06	3.7E-08	1.3E-08	--	--	8.7E-03	3E-04	2E+00	--	5E+01	--	--	--	4E-07	4E-07
Beryllium	5.9	1E-07	3.7E-08	1.3E-08	--	--	8.7E-03	5E-03	4E+00	--	8E+00	4E-05	3E-07	--	1E-08	3E-07
Cadmium	134	1E-05	3.7E-08	1.3E-08	--	--	8.7E-03	1E-03	--	--	6E+00	5E-03	--	--	5E-07	5E-07
Vanadium	845	4E-05	3.7E-08	1.3E-08	--	--	8.7E-03	7E-03	--	--		4E-03	--	--	--	--
Fluoride	10628	--	3.7E-08	--	--	--	--	6E-02	--	--	--	6E-03	--	--	--	--
												Pathway Risk	3E-07	--	1E-06	1E-06
RADIONUCLIDES																
Lead-210+D	357	2E-03	--	--	2.3E+01	3.9E-01	1.6E+04	--	7E-10	2E-10	4E-09	--	6E-06	2E-08	1E-07	6E-06
Radium-226+D	27	1E-03	--	--	2.3E+01	3.9E-01	1.6E+04	--	1E-10	6E-06	3E-09	--	8E-08	6E-05	5E-08	6E-05
Thorium-230	27	1E-03	--	--	2.3E+01	3.9E-01	1.6E+04	--	1E-11	5E-11	3E-08	--	8E-09	6E-10	5E-07	5E-07
Uranium-238+D	24	1E-03	--	--	2.3E+01	3.9E-01	1.6E+04	--	2E-11	5E-08	2E-08	--	1E-08	5E-07	4E-07	9E-07
												Pathway Risk	6E-06	6E-05	1E-06	7E-05

(a) Units are not shown for concentrations, factors, or toxicity values; units are listed in Table B-3, Appendix A, Section 2.0.

BB = Below background

Table B-2b (Continued)
Calculation[a] of Hazards and Risks at Onsite Source Areas
Current Industrial Scenario

	Concentration		Factors (Table B-1)					Reference Doses (RfD) and Slope Factors (SF)				Hazard Quotients and Cancer Risks				
			Oral Intake			External	Inhalation	Non-Cancer	Cancer - Metals/Rads							
			Metals		Radionuclides	Exposure	Intake	RfDs	SFs			Hazard	Cancer Risk			
COC	Source	Air	Non-Cancer	Cancer	Cancer	Cancer	Cancer	Oral RfD	Oral	External	Inhalation	Quotient	Ingestion	External	Inhalation	TOTAL
UNDERFLOW SOLIDS AREA																
Arsenic	245	1E-06	1.6E-07	5.7E-08	--	--	3.8E-02	3E-04	2E+00	--	5E+01	1E-01	2E-05	--	2E-06	3E-05
Beryllium	15	1E-07	1.6E-07	5.7E-08	--	--	3.8E-02	5E-03	4E+00	--	8E+00	5E-04	4E-06	--	5E-08	4E-06
Cadmium	1881	1E-05	1.6E-07	5.7E-08	--	--	3.8E-02	1E-03	--	--	6E+00	3E-01	--	--	2E-06	2E-06
Vanadium	2609	4E-05	1.6E-07	5.7E-08	--	--	3.8E-02	7E-03	--	--	--	6E-02	--	--	--	--
												Pathway Risk	3E-05	--	4E-06	3E-05
RADIONUCLIDES																
Lead-210+D	341	2E-03	--	--	1.0E+02	1.7E+00	6.8E+04	--	7E-10	2E-10	4E-09	--	2E-05	9E-08	6E-07	2E-05
Radium-226+D	41	1E-03	--	--	1.0E+02	1.7E+00	6.8E+04	--	1E-10	6E-06	3E-09	--	5E-07	4E-04	2E-07	4E-04
Thorium-230	45	1E-03	--	--	1.0E+02	1.7E+00	6.8E+04	--	1E-11	5E-11	3E-08	--	6E-08	4E-09	2E-06	2E-06
Uranium-238+D	43	1E-03	--	--	1.0E+02	1.7E+00	6.8E+04	--	2E-11	5E-08	2E-08	--	9E-08	4E-06	2E-06	5E-06
												Pathway Risk	2E-05	4E-04	5E-06	4E-04

(a) Units are not shown for concentrations, factors, or toxicity values; units are listed in Table B-3, Appendix A, Section 2.0.

NOTE: The hazard quotient for arsenic, cadmium, and arsenic, if summed, would be greater than 1.0.

Table B-3
Calculation of Intake Factors and Exposure Factors for the Residential Scenarios

Calculation of Oral Intake Factor (inorganics in soils)
Intake Factor (RME) = (Ingestion Factor x Exposure Frequency)/(Averaging Time x 1E+06 mg/kg)
Intake Factor (AVG) = (Ingestion Rate x Exposure Frequency x Exposure Duration)/(Averaging Time x Body Weight x 1E+06 mg/kg)

Hazard or Risk	Ingestion Factor* (mg-yr/kg-d)	Ingestion Rate (mg/d)	Exposure Frequency (d/yr)	Exposure Duration (yr)	Averaging Time (d)	Body Weight (kg)	Intake Factor (1/d)
Non-cancer (RME)	114		350		10,950		3.6E-06
Cancer (RME)	114		350		25,550		1.6E-06
Non-cancer (AVG)		100	275	9	3,285	70	1.1E-6
Cancer (AVG)		100	275	9	25,550	70	1.4E-07

* Based on integration of child/adult soil intake rates/exposure durations

Calculation of Oral Intake Factor (radionuclides in soils)
Intake Factor (RME) = Ingestion Factor x Exposure Frequency x 1E-03 g/mg
Intake Factor (AVG) = Ingestion Rate x Exposure Frequency x Exposure Duration x 1E-03 g/mg

Hazard or Risk	Ingestion Factor* (mg-yr/d)	Ingestion Rate (mg/d)	Exposure Frequency (d/yr)	Exposure Duration (yr)			Intake Factor (g)
Cancer (RME)	3,600		350				1,260
Cancer (AVG)		100	275	9			248

* Based on integration of child/adult soil intake rates/exposure durations

Calculation of External Exposure Factor (radionuclides)
Exposure Factor = Exposure Duration x (1-Gamma Shielding Factor) x Gamma Exposure Factor
Gamma Exposure Factor = (Hours Exposed/Day) x (Exposure Frequency)/(365 days/yr x 24 hrs/day)

Hazard or Risk	Gamma Shielding Factor	Gamma Exposure Time Factor	Exposure Duration (yr)				Exposure Factor (yr)
Cancer (RME)	0.2	1	30				24
Cancer (AVG)	0.2	1	9				7.2

Table B-3 (Continued)
Calculation of Intake Factors and Exposure Factors
Residential Scenarios

Calculation of Inhalation Intake Factors (inorganics in air)
Intake Factor = (Inhalation Rate x Exposure Frequency x Exposure Duration/(Averaging Time x Body Weight)

Hazard or Risk	Inhalation Rate (m^3/d)	Exposure Frequency (d/yr)	Exposure Duration (yr)	Averaging Time (d)	Body Weight (kg)		Intake Factor (m^3/kg-d)
Non-Cancer (RME)	20	350	30	10,950	70		0.274
Cancer (RME)	20	350	30	25,550	70		0.117
Non-Cancer (AVE)	20	275	9	3,285	70		0.215
Cancer (AVE)	20	275	9	25,550	70		0.028

Calculation of Inhalation Intake Factors (radionuclides in air)
Intake Factor = (Exposure Duration x Exposure Frequency x Inhalation Rate)

Hazard or Risk	Exposure Duration (yr)	Exposure Frequency (d/yr)	Inhalation Rate (m^3/d)				Intake Factor (m^3)
Cancer (RME)	30	350	20				2.1E+05
Cancer (AVE)	9	275	20				5.0E+04

Calculation of Ground Water Intake Factor
Intake Factor = (Ingestion Rate x Exposure Frequency x Exposure Duration)/(Averaging Time x Body Weight)

Hazard or Risk	Water Ingestion Rate (l/d)	Exposure Frequency (d/yr)	Exposure Duration (yr)	Averaging Time (d)	Body Weight (kg)		Intake Factor (l/kg-d)
Non-cancer (RME)	2	350	30	10,950	70		2.7E-02
Cancer (RME)	2	350	30	25,550	70		1.2E-02
Non-cancer (AVG)	1.4	275	9	3285	70		1.5E-02
Cancer (AVG)	1.4	275	9	25,550	70		1.9E-03

Table B-4a
Calculation[a] of Hazards and Risks for the Current Residential Scenario
SOUTHERN

	Concentration		Factors (Table B-3)						RfDs/Slope Factors (Appx A)			Hazard Quotients and Cancer Risks						
			Metals - noncancer		Metals/Rads - cancer		External Exposure Factor					Hazard Quotient		Cancer Risk				
COC	RME	AVG	RME	AVG	RME	AVG	RME	AVG	Noncar-cinogenic	Carcinogenic Metals / Rads		RME	AVG	RME			AVG	
SOIL INGESTION AND EXTERNAL EXPOSURE																		
			Oral	Oral	Oral	Oral			Oral RfD	Oral SF	Ext. SF			Oral	Ext	Total	Oral	Ext	Total
Arsenic	9	5.8	3.6E-06	1.1E-06	1.6E-06	1.4E-07	--	--	3E-04	2E+00	--	1E-01	2E-02	2E-05	--	2E-05	1E-06	--	1E-06
Beryllium	BB	BB	3.6E-06	1.1E-06	1.6E-06	1.4E-07	--	--	5E-03	4E+00	--	--	--	--	--	--	--	--	--
Cadmium	6.4	4.8	3.6E-06	1.1E-06	1.6E-06	1.4E-07	--	--	1E-03	--	--	2E-02	5E-03	--	--	--	--	--	--
Vanadium	40	32	3.6E-06	1.1E-06	1.6E-06	1.4E-07	--	--	7E-03	--	--	2E-02	5E-03	--	--	--	--	--	--
													TOTAL RISK	2E-05	--	2E-05	1E-06	--	1E-06
Radionuclides																			
Pb-210+D	2.9	2.6	--	--	1.3E+03	3.2E+02	2.4E+01	7.2E+00	--	7E-10	2E-10	--	--	2E-06	1E-08	2E-06	5E-07	3E-09	5E-07
Ra-226+D	BB	BB	--	--	1.3E+03	3.2E+02	2.4E+01	7.2E+00	--	1E-10	6E-06	--	--	--	--	--	--	--	--
Th-230	2.1	1.7	--	--	1.3E+03	3.2E+02	2.4E+01	7.2E+00	--	1E-11	5E-11	--	--	3E-08	3E-09	4E-08	7E-09	7E-10	8E-09
U-238+D	2.1	1.7	--	--	1.3E+03	3.2E+02	2.4E+01	7.2E+00	--	2E-11	5E-08	--	--	5E-08	3E-06	3E-06	1E-08	6E-07	6E-07
													TOTAL RISK	2E-06	3E-06	5E-06	6E-07	6E-07	1E-06
AIR																			
					Inhalation					Inh SF									
Arsenic	2.5E-07	2.5E-07			1.2E-01	2.8E-02				5E+01						1E-06			3E-07
Beryllium	6.8E-08	6.8E-08			1.2E-01	2.8E-02				8E+00						7E-08			2E-08
Cadmium	3.3E-06	3.3E-06			1.2E-01	2.8E-02				6E+00						2E-06			6E-07
Vanadium	1.4E-05	1.4E-05			1.2E-01	2.8E-02				--						--			--
															TOTAL RISK	4E-06			9E-07
Radionuclides																			
Pb-210+D	6.6E-04	6.6E-04			2.1E+05	5.0E+04				4E-09						6E-07			1E-07
Ra-226+D	6.2E-04	6.2E-04			2.1E+05	5.0E+04				3E-09						4E-07			9E-08
Th-230	6.5E-04	6.5E-04			2.1E+05	5.0E+04				3E-08						4E-06			9E-07
U-238+D	5.7E-04	5.7E-04			2.1E+05	5.0E+04				2E-08						3E-06			7E-07
															TOTAL RISK	8E-06			2E-06

(a) Units are not shown for concentrations, factors, or toxicity values; units are listed in Table B-3, Appendix A, Section 2.0.

Table B-4b
Calculation[a] of Hazards and Risks for the Current Residential Scenario
WESTERN

	Concentration		Factors (Table B-3)						RfDs/Slope Factors (Appx A)			Hazard Quotients and Cancer Risks							
			Metals - noncancer		Metals/Rads - cancer		External Exposure Factor		Noncar-	Carcinogenic		Hazard Quotient		Cancer Risk					
COC	RME	AVG	RME	AVG	RME	AVG	RME	AVG	cinogenic	Metals / Rads		RME	AVG	RME			AVG		
SOIL INGESTION AND EXTERNAL EXPOSURE																			
			Oral	Oral	Oral	Oral			Oral RfD	Oral SF	Ext. SF			Oral	Ext	Total	Oral	Ext	Total
Arsenic	BB	BB	3.6E-06	1.1E-06	1.6E-06	1.4E-07	--	--	3E-04	2E+00	--	--	--	--	--	--	--	--	--
Beryllium	1.8	BB	3.6E-06	1.1E-06	1.6E-06	1.4E-07	--	--	5E-03	4E+00	--	1E-03	--	1E-05	--	1E-05	--	--	--
Cadmium	11	4.3	3.6E-06	1.1E-06	1.6E-06	1.4E-07	--	--	1E-03	--	--	4E-02	5E-03	--	--	--	--	--	--
Vanadium	43	BB	3.6E-06	1.1E-06	1.6E-06	1.4E-07	--	--	7E-03	--	--	2E-02	--	--	--	--	--	--	--
													TOTAL RISK	1E-05	--	1E-05	--	--	--
Radionuclides																			
Pb-210+D	6.6	4.2	--	--	1.3E+03	3.2E+02	2.4E+01	7.2E+00	--	7E-10	2E-10	--	--	5E-06	3E-08	6E-06	9E-07	5E-09	9E-07
Ra-226+D	BB	BB	--	--	1.3E+03	3.2E+02	2.4E+01	7.2E+00	--	1E-10	6E-06	--	--	--	--	--	--	--	--
Th-230	1.8	BB	--	--	1.3E+03	3.2E+02	2.4E+01	7.2E+00	--	1E-11	5E-11	--	--	3E-08	2E-09	3E-08	--	--	--
U-238+D	BB	BB	--	--	1.3E+03	3.2E+02	2.4E+01	7.2E+00	--	2E-11	5E-08	--	--	--	--	--	--	--	--
													TOTAL RISK	6E-06	3E-08	6E-06	9E-07	5E-09	9E-07
AIR																			
					Inhalation					Inh SF									
Arsenic	7.4E-08	7.4E-08			1.2E-01	2.8E-02				5E+01						4E-07			1E-07
Beryllium	1.6E-08	1.6E-08			1.2E-01	2.8E-02				8E+00						2E-08			4E-09
Cadmium	9.8E-07	9.8E-07			1.2E-01	2.8E-02				6E+00						7E-07			2E-07
Vanadium	3.0E-06	3.0E-06			1.2E-01	2.8E-02				--						--			--
														TOTAL RISK		1E-06			3E-07
Radionuclides																			
Pb-210+D	2.0E-04	2.0E-04			2.1E+05	5.0E+04				4E-09						2E-07			4E-08
Ra-226+D	1.1E-04	1.1E-04			2.1E+05	5.0E+04				3E-09						7E-08			2E-08
Th-230	1.1E-04	1.1E-04			2.1E+05	5.0E+04				3E-08						7E-07			2E-07
U-238+D	1.0E-04	1.0E-04			2.1E+05	5.0E+04				2E-08						5E-07			1E-07
															TOTAL RISK	1E-06			3E-07

(a) Units are not shown for concentrations, factors, or toxicity values; units are listed in Table B-3, Appendix A, Section 2.0.

Table B-4c
Calculation[a] of Hazards and Risks for the Current Residential Scenario
NORTHERN

	Concentration		Factors (Table B-3)						RfDs/Slope Factors (Appx A)			Hazard Quotients and Cancer Risks						
			Metals - noncancer		Metals/Rads - cancer		External Exposure Factor					Hazard Quotient		Cancer Risk				
COC	RME	AVG	RME	AVG	RME	AVG	RME	AVG	Noncar-cinogenic	Carcinogenic Metals / Rads		RME	AVG	RME			AVG	
SOIL INGESTION AND EXTERNAL EXPOSURE																		
			Oral	Oral	Oral	Oral			Oral RfD	Oral SF	Ext. SF			Oral	Ext	Total	Oral	Ext	Total
Arsenic	BB	BB	3.6E-06	1.1E-06	1.6E-06	1.4E-07	--	--	3E-04	2E+00	--	--	--	--	--	--	--	--	--
Beryllium	1.6	1.466667	3.6E-06	1.1E-06	1.6E-06	1.4E-07	--	--	5E-03	4E+00	--	1E-03	3E-04	1E-05	--	1E-05	9E-07	--	9E-07
Cadmium	BB	BB	3.6E-06	1.1E-06	1.6E-06	1.4E-07	--	--	1E-03	--	--	--	--	--	--	--	--	--	--
Vanadium	BB	BB	3.6E-06	1.1E-06	1.6E-06	1.4E-07	--	--	7E-03	--	--	--	--	--	--	--	--	--	--
													TOTAL RISK	1E-05	--	1E-05	9E-07	--	9E-07
Radionuclides																			
Pb-210+D	3.7	2.6	--	--	1.3E+03	3.2E+02	2.4E+01	7.2E+00	--	7E-10	2E-10	--	--	3E-06	1E-08	3E-06	5E-07	3E-09	6E-07
Ra-226+D	BB	BB	--	--	1.3E+03	3.2E+02	2.4E+01	7.2E+00	--	1E-10	6E-06	--	--	--	--	--	--	--	--
Th-230	1.6	BB	--	--	1.3E+03	3.2E+02	2.4E+01	7.2E+00	--	1E-11	5E-11	--	--	3E-08	2E-09	3E-08	--	--	--
U-238+D	BB	BB	--	--	1.3E+03	3.2E+02	2.4E+01	7.2E+00	--	2E-11	5E-08	--	--	--	--	--	--	--	--
													TOTAL RISK	3E-06	2E-08	3E-06	5E-07	3E-09	6E-07
AIR																			
					Inhalation					Inh SF									
Arsenic	9.1E-08	9.1E-08			1.2E-01	2.8E-02				5E+01						5E-07			1E-07
Beryllium	1.2E-08	1.2E-08			1.2E-01	2.8E-02				8E+00						1E-08			3E-09
Cadmium	1.4E-06	1.4E-06			1.2E-01	2.8E-02				6E+00						1E-06			2E-07
Vanadium	1.6E-06	1.6E-06			1.2E-01	2.8E-02				--						--			--
														TOTAL RISK		2E-06			4E-07
Radionuclides																			
Pb-210+D	1.6E-04	1.6E-04			2.1E+05	5.0E+04				4E-09						1E-07			3E-08
Ra-226+D	5.7E-05	5.7E-05			2.1E+05	5.0E+04				3E-09						4E-08			8E-09
Th-230	6.0E-05	6.0E-05			2.1E+05	5.0E+04				3E-08						4E-07			9E-08
U-238+D	5.4E-05	5.4E-05			2.1E+05	5.0E+04				2E-08						3E-07			6E-08
															TOTAL RISK	8E-07			2E-07

(a) Units are not shown for concentrations, factors, or toxicity values; units are listed in Table B-3, Appendix A, Section 2.0.

Table B-4d
Calculation[a] of Hazards and Risks for the Future Southern II Residential Scenario

COC	Concentration		Factors (Table B-3)						RfDs/Slope Factors (Appx A)			Hazard Quotients and Cancer Risks						
			Metals - noncancer		Metals/Rads - cancer		External Exposure Factor					Hazard Quotient		Cancer Risk				
	RME	AVG	RME	AVG	RME	AVG	RME	AVG	Noncar-cinogenic	Carcinogenic Metals / Rads		RME	AVG	RME			AVG	
SOIL INGESTION AND EXTERNAL EXPOSURE																		
			Oral	Oral	Oral	Oral			Oral RfD	Oral SF	Ext. SF			Oral	Ext	Total	Oral	Ext	Total
Arsenic	4.9	BB	3.6E-06	1.1E-06	1.6E-06	1.4E-07	--	--	3E-04	2E+00	--	6E-02	--	1E-05	--	1E-05	--	--	--
Beryllium	1.4	1.3	3.6E-06	1.1E-06	1.6E-06	1.4E-07	--	--	5E-03	4E+00	--	1E-03	3E-04	9E-06	--	9E-06	8E-07	--	8E-07
Cadmium	6.4	3.7	3.6E-06	1.1E-06	1.6E-06	1.4E-07	--	--	1E-03	--	--	2E-02	4E-03	--	--	--	--	--	--
Vanadium	38	BB	3.6E-06	1.1E-06	1.6E-06	1.4E-07	--	--	7E-03	--	--	2E-02	--	--	--	--	--	--	--
													TOTAL RISK	2E-05	--	2E-05	8E-07	--	8E-07
Radionuclides																			
Pb-210+D	2.9	BB	--	--	1.3E+03	3.2E+02	2.4E+01	7.2E+00	--	7E-10	2E-10	--	--	2E-06	1E-08	2E-06	--	--	--
Ra-226+D	BB	BB	--	--	1.3E+03	3.2E+02	2.4E+01	7.2E+00	--	1E-10	6E-06	--	--	--	--	--	--	--	--
Th-230	2.1	1.6	--	--	1.3E+03	3.2E+02	2.4E+01	7.2E+00	--	1E-11	5E-11	--	--	3E-08	3E-09	4E-08	6E-09	6E-10	7E-09
U-238+D	BB	BB	--	--	1.3E+03	3.2E+02	2.4E+01	7.2E+00	--	2E-11	5E-08	--	--	--	--	--	--	--	--
													TOTAL RISK	2E-06	1E-08	2E-06	6E-09	6E-10	7E-09
GROUNDWATER INGESTION - CURRENT DATA																			
												Hazard Quotient							
Cadmium	0.0050	0.0033	2.7E-02	1.2E-02	1.5E-02	1.9E-03			5E-04			3E-01	8E-02						
Fluoride	5.1	2.04	2.7E-02	1.2E-02	1.5E-02	1.9E-03			6E-02			2E+00	4E-01						
Manganese	0.019	0.0080	2.7E-02	1.2E-02	1.5E-02	1.9E-03			5E-03			1E-01	2E-02						
Molybdenum	0.12	0.05	2.7E-02	1.2E-02	1.5E-02	1.9E-03			5E-03			7E-01	1E-01						
Nitrate as N	6.4	3.3	2.7E-02	1.2E-02	1.5E-02	1.9E-03			2E+00			1E-01	2E-02						
Selenium	0.25	0.17	2.7E-02	1.2E-02	1.5E-02	1.9E-03			5E-03			1E+00	4E-01						
GROUNDWATER INGESTION - ASSUMED DATA																			
Cadmium	2.6	--	2.7E-02	1.2E-02	1.5E-02	1.9E-03			5E-04			1E+02	--						
Fluoride	5.0	--	2.7E-02	1.2E-02	1.5E-02	1.9E-03			6E-02			2E+00	--						
Manganese	0.28	--	2.7E-02	1.2E-02	1.5E-02	1.9E-03			5E-03			2E+00	--						
Molybdenum	0.22	--	2.7E-02	1.2E-02	1.5E-02	1.9E-03			5E-03			1E+00	--						
Nitrate as N	11	--	2.7E-02	1.2E-02	1.5E-02	1.9E-03			2E+00			2E-01	--						
Selenium	0.36	--	2.7E-02	1.2E-02	1.5E-02	1.9E-03			5E-03			2E+00	--						

Table B-4d (Continued)
Calculation[a] of Hazards and Risks for the Future Southern II Residential Scenario

	Concentration		Factors (Table B-3)						RfDs/Slope Factors (Appx A)		Hazard Quotients and Cancer Risks							
			Metals - noncancer		Metals/Rads - cancer		External Exposure Factor											
COC	RME	AVG	RME	AVG	RME	AVG	RME	AVG	Noncar-cinogenic	Carcinogenic Metals / Rads	Hazard Quotient RME	Hazard Quotient AVG	Cancer Risk RME			Cancer Risk AVG		
AIR																		
					Inhalation					Inh SF								
Arsenic	8.6E-08	8.6E-08			1.2E-01	2.8E-02				5E+01					5E-07			1E-07
Beryllium	1.9E-08	1.9E-08			1.2E-01	2.8E-02				8E+00					2E-08			4E-09
Cadmium	1.1E-06	1.1E-06			1.2E-01	2.8E-02				6E+00					8E-07			2E-07
Vanadium	2.1E-06	2.1E-06			1.2E-01	2.8E-02				--					--			--
														TOTAL RISK	1E-06			3E-07
Radionuclides																		
Pb-210+D	3.0E-04	3.0E-04			2.1E+05	5.0E+04				4E-09					2E-07			6E-08
Ra-226+D	6.8E-05	6.8E-05			2.1E+05	5.0E+04				3E-09					4E-08			1E-08
Th-230	6.9E-05	6.9E-05			2.1E+05	5.0E+04				3E-08					4E-07			1E-07
U-238+D	6.7E-05	6.7E-05			2.1E+05	5.0E+04				2E-08					3E-07			8E-08
														TOTAL RISK	1E-06			2E-07

(a) Units are not shown for concentrations, factors, or toxicity values; units are listed in Table B-3, Appendix A, Section 2.0.
BB = Below background UCL Concentrations.

Table B-4e
Calculation[a] of Hazards and Risks for the Future Northern I Residential Scenario

COC	Concentration		Factors (Table B-3)						RfDs/Slope Factors (Appx A)			Hazard Quotients and Cancer Risks						
			Metals - noncancer		Metals/Rads - cancer		External Exposure Factor		Noncar-cinogenic	Carcinogenic Metals / Rads		Hazard Quotient		Cancer Risk				
	RME	AVG	RME	AVG	RME	AVG	RME	AVG				RME	AVG	RME			AVG		
SOIL INGESTION AND EXTERNAL EXPOSURE																		
			Oral	Oral	Oral	Oral			Oral RfD	Oral SF	Ext. SF			Oral	Ext	Total	Oral	Ext	Total
Arsenic	34	22	3.6E-06	1.1E-06	1.6E-06	1.4E-07	--	--	3E-04	2E+00	--	4E-01	8E-02	9E-05	--	9E-05	5E-06	--	5E-06
Beryllium	3.7	3.1	3.6E-06	1.1E-06	1.6E-06	1.4E-07	--	--	5E-03	4E+00	--	3E-03	7E-04	2E-05	--	2E-05	2E-06	--	2E-06
Cadmium	153	101	3.6E-06	1.1E-06	1.6E-06	1.4E-07	--	--	1E-03	--	--	6E-01	1E-01	--	--	--	--	--	--
Vanadium	371	272	3.6E-06	1.1E-06	1.6E-06	1.4E-07	--	--	7E-03	--	--	2E-01	4E-02	--	--	--	--	--	--
													TOTAL RISK	1E-04	--	1E-04	7E-06	--	7E-06
Radionuclides																			
Pb-210+D	65	45	--	--	1.3E+03	3.2E+02	2.4E+01	7.2E+00	--	7E-10	2E-10	--	--	5E-05	2E-07	5E-05	9E-06	5E-08	9E-06
Ra-226+D	13	9.4	--	--	1.3E+03	3.2E+02	2.4E+01	7.2E+00	--	1E-10	6E-06	--	--	2E-06	2E-03	2E-03	4E-07	4E-04	4E-04
Th-230	12	8.5	--	--	1.3E+03	3.2E+02	2.4E+01	7.2E+00	--	1E-11	5E-11	--	--	2E-07	2E-08	2E-07	3E-08	3E-09	4E-08
U-238+D	11	8.2	--	--	1.3E+03	3.2E+02	2.4E+01	7.2E+00	--	2E-11	5E-08	--	--	3E-07	1E-05	1E-05	5E-08	3E-06	3E-06
													TOTAL RISK	6E-05	2E-03	2E-03	1E-05	4E-04	4E-04
AIR																			
					Inhalation					Inh SF									
Arsenic	9.3E-07	9.3E-07			1.2E-01	2.8E-02				5E+01						5E-06			1E-06
Beryllium	1.5E-07	1.5E-07			1.2E-01	2.8E-02				8E+00						1E-07			3E-08
Cadmium	1.2E-05	1.2E-05			1.2E-01	2.8E-02				6E+00						8E-06			2E-06
Vanadium	2.0E-05	2.0E-05			1.2E-01	2.8E-02				--						--			--
															TOTAL RISK	1E-05			3E-06
Radionuclides																			
Pb-210+D	2.9E-03	2.9E-03			2.1E+05	5.0E+04				4E-09						2E-06			6E-07
Ra-226+D	3.9E-04	3.9E-04			2.1E+05	5.0E+04				3E-09						2E-07			6E-08
Th-230	4.4E-04	4.4E-04			2.1E+05	5.0E+04				3E-08						3E-06			6E-07
U-238+D	4.4E-04	4.4E-04			2.1E+05	5.0E+04				2E-08						2E-06			5E-07
															TOTAL RISK	8E-06			2E-06

(a) Units are not shown for concentrations, factors, or toxicity values; units are listed in Table B-3, Appendix A, Section 2.0.

Table B-4f

Calculation[a] of Hazards and Risks for the Future Northern II Residential Scenario

	Concentration		Factors (Table B-3)						RfDs/Slope Factors (Appx A)			Hazard Quotients and Cancer Risks						
			Metals - noncancer		Metals/Rads - cancer		External Exposure Factor					Hazard Quotient		Cancer Risk				
COC	RME	AVG	RME	AVG	RME	AVG	RME	AVG	Noncar-cinogenic	Carcinogenic Metals / Rads		RME	AVG	RME			AVG		
SOIL INGESTION AND EXTERNAL EXPOSURE																		
			Oral	Oral	Oral	Oral			Oral RfD	Oral SF	Ext. SF			Oral	Ext	Total	Oral	Ext	Total
Arsenic	10	7.5	3.6E-06	1.1E-06	1.6E-06	1.4E-07	--	--	3E-04	2E+00	--	1E-01	3E-02	3E-05	--	3E-05	2E-06	--	2E-06
Beryllium	1.4	1.2	3.6E-06	1.1E-06	1.6E-06	1.4E-07	--	--	5E-03	4E+00	--	1E-03	3E-04	9E-06	--	9E-06	7E-07	--	7E-07
Cadmium	16	9.4	3.6E-06	1.1E-06	1.6E-06	1.4E-07	--	--	1E-03	--	--	6E-02	1E-02	--	--	--	--	--	--
Vanadium	68	42	3.6E-06	1.1E-06	1.6E-06	1.4E-07	--	--	7E-03	--	--	4E-02	6E-03	--	--	--	--	--	--
													TOTAL RISK	4E-05	--	4E-05	3E-06	--	3E-06
Radionuclides																			
Pb-210+D	6.9	4.2	--	--	1.3E+03	3.2E+02	2.4E+01	7.2E+00	--	7E-10	2E-10	--	--	6E-06	3E-08	6E-06	9E-07	5E-09	9E-07
Ra-226+D	2.5	1.9	--	--	1.3E+03	3.2E+02	2.4E+01	7.2E+00	--	1E-10	6E-06	--	--	4E-07	4E-04	4E-04	7E-08	8E-05	8E-05
Th-230	3.1	2.0	--	--	1.3E+03	3.2E+02	2.4E+01	7.2E+00	--	1E-11	5E-11	--	--	5E-08	4E-09	5E-08	8E-09	8E-10	9E-09
U-238+D	2.8	2.2	--	--	1.3E+03	3.2E+02	2.4E+01	7.2E+00	--	2E-11	5E-08	--	--	7E-08	3E-06	3E-06	1E-08	8E-07	8E-07
													TOTAL RISK	6E-06	4E-04	4E-04	1E-06	8E-05	9E-05
AIR																			
					Inhalation					Inh SF									
Arsenic	4.0E-08	4.0E-08			1.2E-01	2.8E-02				5E+01						2E-07			5E-08
Beryllium	6.7E-09	6.7E-09			1.2E-01	2.8E-02				8E+00						7E-09			2E-09
Cadmium	5.1E-07	5.1E-07			1.2E-01	2.8E-02				6E+00						4E-07			9E-08
Vanadium	1.0E-06	1.0E-06			1.2E-01	2.8E-02				--						--			--
															TOTAL RISK	6E-07			1E-07
Radionuclides																			
Pb-210+D	1.5E-04	1.5E-04			2.1E+05	5.0E+04				4E-09						1E-07			3E-08
Ra-226+D	2.3E-05	2.3E-05			2.1E+05	5.0E+04				3E-09						1E-08			3E-09
Th-230	2.5E-05	2.5E-05			2.1E+05	5.0E+04				3E-08						2E-07			4E-08
U-238+D	2.4E-05	2.4E-05			2.1E+05	5.0E+04				2E-08						1E-07			3E-08
															TOTAL RISK	4E-07			1E-07

(a) Units are not shown for concentrations, factors, or toxicity values; units are listed in Table B-3, Appendix A, Section 2.0.

Table B-4g

Calculation[a] of Hazards and Risks for the Future Southern I Residential Scenario

	Concentration		Factors (Table B-3)						RfDs/Slope Factors (Appx A)			Hazard Quotients and Cancer Risks						
			Metals - noncancer		Metals/Rads - cancer		External Exposure Factor					Hazard Quotient		Cancer Risk				
														RME			AVG		
COC	RME	AVG	RME	AVG	RME	AVG	RME	AVG	Noncar-cinogenic	Carcinogenic Metals / Rads		RME	AVG	Oral	Ext	Total	Oral	Ext	Total
SOIL INGESTION AND EXTERNAL EXPOSURE																			
			Oral	Oral	Oral	Oral			Oral RfD	Oral SF	Ext. SF								
Arsenic	10	8.6	3.6E-06	1.1E-06	1.6E-06	1.4E-07	--	--	3E-04	2E+00	--	1E-01	3E-02	3E-05	--	3E-05	2E-06	--	2E-06
Beryllium	4	2.9	3.6E-06	1.1E-06	1.6E-06	1.4E-07	--	--	5E-03	4E+00	--	3E-03	6E-04	3E-05	--	3E-05	2E-06	--	2E-06
Cadmium	68	37	3.6E-06	1.1E-06	1.6E-06	1.4E-07	--	--	1E-03	--	--	2E-01	4E-02	--	--	--	--	--	--
Vanadium	324	191	3.6E-06	1.1E-06	1.6E-06	1.4E-07	--	--	7E-03	--	--	2E-01	3E-02	--	--	--	--	--	--
													TOTAL RISK	5E-05	--	5E-05	4E-06	--	4E-06
Radionuclides																			
Pb-210+D	24	13	--	--	1.3E+03	3.2E+02	2.4E+01	7.2E+00	--	7E-10	2E-10	--	--	2E-05	9E-08	2E-05	3E-06	1E-08	3E-06
Ra-226+D	12	7.4	--	--	1.3E+03	3.2E+02	2.4E+01	7.2E+00	--	1E-10	6E-06	--	--	2E-06	2E-03	2E-03	3E-07	3E-04	3E-04
Th-230	16	9.0	--	--	1.3E+03	3.2E+02	2.4E+01	7.2E+00	--	1E-11	5E-11	--	--	3E-07	2E-08	3E-07	4E-08	3E-09	4E-08
U-238+D	10	6.9	--	--	1.3E+03	3.2E+02	2.4E+01	7.2E+00	--	2E-11	5E-08	--	--	3E-07	1E-05	1E-05	4E-08	3E-06	3E-06
													TOTAL RISK	2E-05	2E-03	2E-03	3E-06	3E-04	3E-04
AIR																			
					Inhalation					Inh SF									
Arsenic	7.8E-08	7.8E-08			1.2E-01	2.8E-02				5E+01						5E-07			1E-07
Beryllium	1.8E-08	1.8E-08			1.2E-01	2.8E-02				8E+00						2E-08			4E-09
Cadmium	1.0E-06	1.0E-06			1.2E-01	2.8E-02				6E+00						7E-07			2E-07
Vanadium	1.9E-06	1.9E-06			1.2E-01	2.8E-02				--						--			--
															TOTAL RISK	1E-06			3E-07
Radionuclides																			
Pb-210+D	2.6E-04	2.6E-04			2.1E+05	5.0E+04				4E-09						2E-07			5E-08
Ra-226+D	6.3E-05	6.3E-05			2.1E+05	5.0E+04				3E-09						4E-08			9E-09
Th-230	6.5E-05	6.5E-05			2.1E+05	5.0E+04				3E-08						4E-07			9E-08
U-238+D	6.2E-05	6.2E-05			2.1E+05	5.0E+04				2E-08						3E-07			7E-08
															TOTAL RISK	1E-06			2E-07

(a) Units are not shown for concentrations, factors, or toxicity values; units are listed in Table B-3, Appendix A, Section 2.0.
(b) Currently unoccupied home.

Appendix C

Calculation of Background Risks

Table C-1
Calculation of Hazards and Risks at Background
Future Industrial Scenario

		Intake Factors (Table B-1)			Reference Doses (RfD) and Slope Factors (SF)			Hazard Quotients and Cancer Risks			
	Concentration	Oral Intake		External Exposure	Non-cancer RfD	Cancer - Metals/Rads SFs		Hazard	Cancer Risk		
COC	Source	Non-Cancer	Cancer	Cancer	Oral	Oral	External	Quotient	Ingestion	External	TOTAL
Arsenic	4.4	2.9E-07	1.0E-07	--	3E-04	2E+00	--	4E-03	8E-07	--	8E-07
Beryllium	1.2	2.9E-07	1.0E-07	--	5E-03	4E+00	--	7E-05	5E-07	--	5E-07
Cadmium	2.1	2.9E-07	1.0E-07	--	1E-03	--	--	6E-04	--	--	--
Vanadium	25.8	2.9E-07	1.0E-07	--	7E-03	--	--	1E-03	--	--	--
								Pathway Risk	1E-06	--	1E-06
RADIONUCLIDES											
Lead-210+D	2.5	--	1.9E+02	6.0	--	7E-10	2E-10	--	3E-07	2E-09	3E-07
Radium-226+D	1.9	--	1.9E+02	6.0	--	1E-10	6E-06	--	4E-08	7E-05	7E-05
Thorium-230	1.4	--	1.9E+02	6.0	--	1E-11	5E-11	--	3E-09	5E-10	4E-09
Uranium-238+D	1.7	--	1.9E+02	6.0	--	2E-11	5E-08	--	6E-09	5E-07	5E-07
								Pathway Risk	4E-07	7E-05	7E-05

(a) Units are not shown for concentrations, factors, or toxicity values; units are listed in Table B-3, Appendix A, Section 2.0.

Table C-2
Risks at Background
Industrial Scenarios

BACKGROUND RISKS (Future Industrial)			
	Oral	External	Total
Arsenic	8E-07	--	8E-07
Beryllium	5E-07	--	5E-07
		TOTAL	1E-06
Radionuclides			
Lead-210+D	3E-07	2E-09	3E-07
Radium-226+D	4E-08	7E-05	7E-05
Thorium-230	3E-09	5E-10	4E-09
Uranium-238+D	6E-09	5E-07	5E-07
		TOTAL	7E-05
CURRENT INDUSTRIAL BACKGROUND RISKS (Scaled from Future Industrial)			
	Future Total	Scaler	Current Total
BAGHOUSE DUST AREA			
Metals	1E-06	0.19	3E-07
Radionuclides	7E-05	0.18	1E-05
NODULES			
Metals	1E-06	0.19	3E-07
Radionuclides	7E-05	0.18	1E-05
SLAG			
Metals	1E-06	0.50	7E-07
Radionuclides	7E-05	0.26	2E-05
ROAD DUSTS			
Metals	1E-06	0.48	6E-07
Radionuclides	7E-05	0.25	2E-05
TREATER DUST AREA			
Metals	1E-06	0.13	2E-07
Radionuclides	7E-05	0.07	5E-06
UNDERFLOW SOLIDS AREA			
Metals	1E-06	0.54	7E-07
Radionuclides	7E-05	0.28	2E-05

Table C-3
Calculation of RME Hazards and Risks at Background
Residential Scenarios

COC	Soil Concentration	Factors (Table B-3)			Reference Doses and Slope Factors			Hazard Quotients and Cancer Risks			
		Oral Intake		External Exposure	Non-Cancer RfDs	Cancer -Metals/Rads SFs		Hazard	Cancer Risk		
		Non-cancer	Cancer	Cancer	Oral RfD	Oral	External	Quotient	Oral	External	TOTAL
Arsenic	4.4	3.6E-06	1.6E-06	--	3E-04	2E+00	--	5E-02	1E-05	--	1E-05
Beryllium	1.2	3.6E-06	1.6E-06	--	5E-03	4E+00	--	9E-04	8E-06	--	8E-06
								Pathway Risk	2E-05	--	2E-05
Radionuclides											
Lead-210	2.5	--	1.3E+03	2.4E+01	--	7E-10	2E-10	--	2E-06	1E-08	2E-06
Radium-226	1.9	--	1.3E+03	2.4E+01	--	1E-10	6E-06	--	3E-07	3E-04	3E-04
Thorium-230	1.4	--	1.3E+03	2.4E+01	--	1E-11	5E-11	--	2E-08	2E-09	2E-08
Uranium-238+D	1.7	--	1.3E+03	2.4E+01	--	2E-11	5E-08	--	4E-08	2E-06	2E-06
								Pathway Risk	2E-06	3E-04	3E-04

(a) Units are not shown for concentrations, factors, or toxicity values; units are listed in Table B-3, Appendix A, Section 2.0.

Appendix D

Toxicological Profiles

ARSENIC

CAS No: 7440-38-2
Synonyms: gray arsenic, metallic arsenic

Physico-chemical Characteristics[1]

Description: Arsenic (As) is a gray, shiny, brittle, rhombohedral metal present ubiquitously in the earth's crust (5 mg/kg).

Criteria and Standards[2]

Safe Drinking Water Act

Maximum Contaminant Level Goal, MCLG	0.05 mg/L
Maximum Contaminant Level, MCL	0.05 mg/L (interim, 1980)

Clean Water Act

Ambient Water Quality Criteria

Human Health	
a. Water and Fish Consumption	2.2E-3 μg/L
b. Fish Consumption Only	1.75E-2 μg/L
Aquatic Organisms	
1. Fresh Water	
a. Acute	3.6 μg/L
b. Chronic	1.9E+2 μg/L
2. Marine	
a. Acute	6.9E+1 μg/L
b. Chronic	3.6E+1 μg/L

NIOSH Recommended Standard (air)	2 $\mu g/m^3$
OSHA Standard (air)	500 $\mu g/m^3$
ACGIH (Threshold Limit Value)	200 $\mu g/m^3$

Environmental Transport and Fate

Arsenic is generally quite mobile in the environment. Trivalent (+3) and pentavalent (+5) forms of arsenic are inter-convertible. Formation of organic complexes of arsenic results in its high mobility and recycling particularly in the aquatic environment. Volatilization of arsenic and its conversion to highly active arsine or methylarsine forms in the presence of highly reducing conditions is an important fate process in the aquatic environment. Biotransformion of arsenic to organic forms is performed by a number of prokaryotic species present in the environment.

General Toxicity

Arsenic demonstrates a high acute as well as chronic toxicity. Systemic chronic poisoning is primarily characterized by skin lesions such as dermatoses, which may include eruptions, pigmentations, or hypekeratosis, that may ultimately lead to the development of skin cancer. Reports of abnormal electrocardiograms and peripheral vascular disturbances with gangrene of the extremities (Blackfoot disease) exist following chronic exposure to arsenic. Hematological changes following arsenic exposure are characterized by anemia and leukopenia.

Common acute symptoms include gastritis, fever, insomnia, anorexia, swollen liver, and cardiac dysfunction. Peripheral nervous disturbances primarily are of sensory type and is frequently encountered among individuals surviving arsenic poisoning.

Chronic symptoms of toxicity generally involve the skin, mucous membranes, lungs, gastrointestinal and nervous system. Effects on the circulatory system and liver is sporadic. Several epidemiological studies have indicated a close association of exposure to inorganic arsenicals and various forms of skin diseases. Hyperkeratosis, warts and melanosis of the skin are the most commonly observed lesions following chronic arsenic exposure. Chronic arsenical poisoning is known to cause peripheral neuritis affecting mainly the upper and lower extremities.

The critical effects upon which the reference dose is based are hyperpigmentation, keratosis and possible vascular complications. These were determined in a chronic human exposure study of arsenic-contaminated water consumption. Although the database is large ($>40{,}000$ people were studied), the doses were not well characterized and other possible contaminants were not examined. Therefore, the confidence in this reference dose is medium.

It has been demonstrated that human blood arsenic levels do not increase until daily consumption of arsenic exceeds μg, or approximately 120 μg/l.

Arsenic compounds have been reported to be teratogenic, fetotoxic and embryotoxic in several animal species. An increased incidence of multiple malformations among children born to women occupationally exposed to arsenic has also been reported.

Evidence of Carcinogenicity

Animal Carcinogenicity data: Experimental data on the effects of arsenic on carcinogenesis are not consistent for various chemical forms of arsenic and routes of administration (IARC 1980). However, there are some data to indicate that arsenic is a carcinogen in experimental animals, if retention time in the lung can be increased. Following an intratracheal instillation, arsenic trioxide was found to enhance incidence of tumorigenesis of the respiratory tract in hamsters. Similarly a high incidence of lung carcinoma was reported in rats following a single intratracheal instillation of a pesticide mixture containing calcium arsenates (IARC 1987). Arsenic was reported to promote Diethyl nitrosamine-induced renal tumors in rats (Kroes et al., 1974).

Sodium arsenic has been shown to transform Syrian hamster embryo cells (Dipaolo and Casto, 1979) and to produce SCE in DON cells, CHO cells and human peripheral lymphocytes exposed in vitro (Wan et al., 1982; Ohno et al., 1982; Larramendy et al., 1981; Andersen, 1983; Crossen, 1983). While arsenic compounds have not been shown to mutate bacterial strains, it produces preferential killing of repair deficient strains (Rossman, 1981).

Human Carcinogenicity data: Studies of smelter worker populations (Tacoma, WA; Magma, UT; Anaconda, MT; Ronnskar, Sweden; Saganoseki-Machii, Japan) have all found an association between occupational arsenic exposure and lung cancer mortality (Enterline and Marsh, 1982; Lee-Feldstein, 1983; Axelson et al., 1978; Tokudome and Kuratsune, 1976; Rencher et al., 1977). Both proportionate mortality and cohort studies of pesticide manufacturing workers have shown excess of lung cancer deaths among exposed persons (Ott et al., 1974; Mabuchi et al., 1979). One study of a population residing near a pesticide manufacturing plant revealed that these residents were also at an excess risk of lung cancer (Matanoski et al., 1981).

A cross-sectional study of 40,000 Taiwanese exposed to arsenic in drinking water found significant excess skin cancer prevalence by comparison to 7500 residents of Taiwan and Matsu who consumed relatively arsenic-free water (Tseng, 1977). Arsenic-induced skin cancer has also been attributed to water supplies in Chile, Argentina and Mexico (Borgono and Greiber, 1972; Bergoglio, 1964; Cebrian et al., 1983). No excess skin cancer incidence has been observed in U.S. residents consuming relatively high levels of arsenic in drinking water (Morton et al., 1976; Southwick et al., 1981). These U.S. studies, however, are not inconsistent with the existing findings from the foreign populations. The statistical powers of the U.S. studies are considered to be inadequate because of the small sample size.

A study of the population living in the same area of Taiwan studied by Tseng (1977), where arsenic contamination of the water supply was endemic, found significantly elevated standard mortality ratios for cancer of the bladder, lung, liver, kidney, skin and colon. A case control study of bladder, liver and lung cancer cases in the endemic area found a significant association with arsenic exposure that was dose-related. The association of arsenic ingestion and cancer of various internal organs has also been cited in a number of case reports.

Carcinogenic Risk Assessment

The EPA Risk Assessment Forum has, recently completed a reassessment of the carcinogenicity risk associated with ingestion of inorganic arsenic. The report is based on the linearized multistage model and a maximum likelihood approach was utilized together with the Weibull model. The report concluded, that the most appropriate basis for an oral estimate was the study by Tseng et al. (1977), which reported increased prevalence of skin cancers in humans as a consequence of arsenic exposure in drinking water. Based on this study a unit risk of 5.0E-05/μg/L was proposed, which is a magnitude lower than the previous estimate (carcinogenic slope factor of 1.75). However, it should be noted, that even when applying this lower estimate, practically no drinking water consumed in the U.S. will fulfill the 1.0E-06 risk goal.

The uncertainties associated with ingested inorganic arsenic are such that estimates could be modified downwards as much as an order of magnitude, relative to risk estimates associated with most other carcinogens (IRIS, 1988).

The EPA estimates of a slope factor of 5.0E+01 $(mg/kg/day)^{-1}$ was based on a unit risk of 4.3E-03/$\mu g/m^3$ (calculated assuming a 70 kg human body weight, 20 m^3 air inhaled/day and a 30% absorption of inhaled arsenic) for inhalation exposure.

[1] Physico-chemical data was obtained from The Merck Index (10th Edition), Merck and Co., Rayway. 1983.

[2] Criteria and Standards information was obtained from the Integrated Risk Information System (IRIS), a new on-line file of EPA available with the TOXNET system as of February 1994.

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BERYLLIUM

CAS No: 7440-47-7
Synonyms: Glucinium

Physico-chemical Characteristics[1]

Description: Beryllium is a gray metal with a natural abundance of 2 to 10 mg/kg. Chemical properties of beryllium are similar to that of aluminum.

Criteria and Standards[2]

The available data on Beryllium are not adequate for establishing criteria. However, EPA has reported the available lowest toxic doses for aquatic organisms.

Safe Drinking Water Act

Maximum Contaminant Level Goal, MCLG	0 mg/L (proposed, 1990)
Maximum Contaminant Level, MCL	1 μg/L (proposed, 1990)

Clean Water Act

Ambient Water Quality Criteria

Human Health	
a. Water & Fish Consumption	6.8E-3 μg/L
b. Fish Consumption only	1.17E-1 μg/L
Aquatic Organisms	
1. Fresh Water	
a. Acute	1.3E+2 μg/L
b. Chronic	5.3E+0 μg/L
2. Marine	
a. Acute	N/A
b. Chronic	N/A

Environmental Transport and Fate

Most common beryllium compounds are readily soluble in water. However, in water, soluble beryllium salts are hydrolyzed to form less soluble beryllium hydroxide. Although little information concerning adsorption of beryllium is available, based on its geochemical similarity to aluminum it is expected to be adsorbed or complexed onto clay mineral surfaces. Transport of beryllium via air is generally in the form of particulates.

Metabolism

Beryllium is not well absorbed (1% absorption may be maximum in experimental animals) when administered by any route. Following inhalation exposure, beryllium is mobilized in the blood stream as a colloidal beryllium phosphate and soluble beryllium-citrate complex that is deposited in the bone or excreted in the urine. The colloidal portion is deposited in the liver, spleen or bone marrow.

General Toxicity

There is equivocal evidence linking occupational exposure to beryllium and lung cancer. Experimental animals exposed to beryllium by inhalation have developed carcinoma of the lung and bone marrow. Acute respiratory effects due to beryllium exposure include rhinitis, pharyngitis, tracheobronchitis, and acute pneumonitis.

Chronic skin lesions sometimes appear after a long latent period in conjugation with the chronic pulmonary aspect of the disease. Dermal exposure to soluble beryllium compounds can cause contact dermatitis. Ocular effects include conjunctivitis from splash burns or in association with contact dermatitis.

The most common symptoms of chronic beryllium exposure are granulomatous lung inflammation, with accompanying cough, chest pain, and general weakness. Systemic effects include right heart enlargement with accompanying cardiac failure, liver and spleen enlargement, cyanosis, digital clubbing, and kidney stone development.

The oral RfD (NOAEL 0.54 mg/kg bw/day) is based on the studies by Schroeder and Mitchner (1975) (IRIS, 1990). Fifty-two weanling Long-Evans rats of each sex received 0 or 5 ppm beryllium (as BeSO4, beryllium sulfate) in drinking water. Exposure was for the lifetime of the animals. At natural death the rats were dissected and gross and microscopic changes were noted in heart, kidney, liver, and spleen. There were no effects of treatment on these organs or on life span, urinalysis, serum glucose, cholesterol, and uric acid, or on numbers of tumors. Male rats experienced decreased growth rates from 2 to 6 months of age.

Similar studies were carried out on Swiss (CD strain) mice at doses of approximately 0.95 mg/kg/day (Schroeder and Mitchner, 1975). Female animals showed decreased body weight compared with untreated mice at 6 of 8 intervals. Male mice exhibited slight increases in body weight. These effects were not considered adverse, therefore, 0.95 mg/kg/day is considered a NOAEL.

A reference dose for chronic inhalation exposure (RfDi) is not available at present (IRIS, 1990).

Uncertainty and modifying factors for the oral RfD is 100 and 1 respectively. The uncertainty factor of 100 reflects a factor of 10 each for inter species conversion and for the protection of sensitive human sub-populations. It is important to note that this RfD is limited to soluble beryllium salts only.

Confidence in the studies determining the oral RfD is rated as low because only one dose level was administered. Although numerous inhalation investigations and a supporting chronic oral bioassay in mice exist, along with the work by Morgareidge et al. (1975) which indicates that a higher dose level might be a NOEL, these studies are considered as low to medium quality; thus, the data base is given a low confidence rating. The overall confidence in the RfD is low, reflecting the need for more toxicity data by the oral route.

Evidence of Carcinogenicity

Animal Carcinogenicity Data: Evidence of beryllium carcinogenicity from animal studies are considered sufficient. Based on the evidence for induction of tumors by a variety of beryllium compounds in male and female monkeys and in several strains of rats of both sexes, via inhalation and intratracheal instillation, and the induction of osteosarcomas in rabbits by intravenous or intramedullary injection in multiple studies.

Osteogenic sarcomas were induced in rabbits by intravenous injection of beryllium compounds in at least 12 different studies and by intramedullary injection in at least four studies (U.S. EPA, 1987). Bone tumors were induced by beryllium oxide, zinc beryllium silicate, beryllium phosphate, beryllium silicate and beryllium metal. Positive results, however, were reported in mice injected with zinc beryllium silicate, although the numbers were not listed (Cloudman et al., 1949). The sarcomas were generally reported to be quite malignant and metastasized to other organs.

Lung tumors, primarily adenomas and adenocarcinomas, have been induced via the inhalation route in both male and female Sprague-Dawley rats during exposure periods of up to 72 weeks by beryllium sulfate (Reeves et al., 1967), by beryllium phosphate in both male and female Wistar rats by beryllium phosphate and zinc beryllium silicate (Schepers, 1961), and in male Charles River CR-CD rats by beryl ore (Wagner et al., 1969).

Tumors were also induced by intratracheal instillation of metallic beryllium, beryllium-aluminum alloys and beryllium oxide in both Wistar rats and rhesus monkeys. Adenomas, adenocarcinomas and malignant lymphomas were seen in the lungs, with lymphosarcomas and fibrosarcomas present at extra pulmonary sites (Groth et al., 1980; Ishinishi et al., 1980). Human Carcinogenicity Data: Human carcinogenicity data on beryllium is considered inadequate. Although several reports exist of increases in the cancer incidence in occupational settings, most of these studies have not taken a variety of possible confounding factors into account. For instance in a study of workers from a beryllium processing plant, and several studies of workers from this plant combined with workers from other beryllium plants, a statistically significant increased incidence of lung cancer (IRIS, 1990, Bayliss and Wagoner, 1977; Mancuso, 1970,

1979, 1980) was reported. However, adjustments were not made for smoking in these studies, and all were limited in their ability to detect a possible increased incidence of lung cancer because of methodological constraints and deficiencies.

Carcinogenicity Risk Assessment

U.S. EPA classifies beryllium as a B2 carcinogen meaning a probable human carcinogen. Weight-of-evidence was based on the observations that beryllium has been shown to induce lung cancer via inhalation in rats and monkeys and to induce osteosarcomas in rabbits via intravenous or intramedullary injection. Human epidemiology studies are considered to be inadequate.

Quantitative estimates of carcinogenic risks from oral exposure (oral slope factor) is 4.3 per(mg/kg)/day. Drinking water Unit risk is estimated at 1.2E-4 per (μg/L). However, it is important to note that the unit risk should not be used if the water concentration exceeds 8.3E+1 μg/L, since above this concentration the slope factor may differ from that stated.

The level of confidence (oral exposure) may be considered limited due to high mortality and unspecified type and site of the tumors and has only one non-zero dose group. Further, the estimate is based on a study which did not show a significant increase in tumorigenic response.

Quantitative estimates of carcinogenic risks from inhalation exposure (inhalation slope factor) is 8.4E+0 per(mg/kg)/day (IRIS, 1990). Inhalation unit risk is estimated at 2.4E-3 per ($\mu g/m^3$). Both the risk estimates are based on relative risk extrapolation method. It is relevant to note that despite several limitations, human data were used to quantify inhalation exposure. Humans are most likely to be exposed by inhalation to beryllium oxide, rather than other beryllium salts. Animal studies by inhalation of beryllium oxide have utilized intratracheal instillation, rather than general inhalation exposure.

The estimates of exposure levels and duration are somewhat uncertain in these studies. While a quantitative assessment based on several animal studies resulted in a similar estimate of risk (which probably increases the confidence), the quality of the available data is considered poor since they lacked multi-dosed studies and adequate controls.

[1] Physico-chemical data was obtained from The Merck Index (10th Edition), Merck and Co, Rahway. 1983.

[2] Criteria and Standards information was obtained from the Integrated Risk Information System (IRIS), a new on-line file of EPA available with the TOXNET system as of February 1994.

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CADMIUM

CAS No: 7440-43-9

Physico-chemical Characteristics[1]

Description: Cadmium is a soft, bluish white lustrous metal with a natural abundance of approximately 0.2 mg/kg in the earth's crust.

Criteria and Standards[2]

Safe Drinking Water Act

Maximum Contaminant Level Goal, MCLG	0.005 mg/L (final, 1991)
Maximum Contaminant Level, MCL	0.005 mg/L (final, 1991)

Clean Water Act

Ambient Water Quality Criteria

Human Health	
a. Water and Fish Consumption	1E+1 μg/L
b. Fish Consumption Only	
Aquatic Organisms	
1. Fresh Water	
a. Acute	3.9E+0 μg/L
b. Chronic	1.1E+0 μg/L
2. Marine	
a. Acute	4.3E+1 μg/L
b. Chronic	9.3E+0 μg/L

NIOSH Recommended Standard (air)	Occupational Carcinogen
OSHA Standard (air)	5 $\mu g/m^3$ TWA
ACGIH (Threshold Limit Value)	50 $\mu g/m^3$ TWA

Environmental Transport and Fate

Cadmium is relatively mobile in the aquatic environment compared to other heavy metals (U.S. EPA, 1979). Complexation with organic materials appears to be the mechanisms of removal of cadmium from aqueous media. Mobility of cadmium transport in the soil is not clearly known. However, cadmium uptake by plants is considered significant.

General Toxicity

Approximately 6 to 10 percent of ingested cadmium is absorbed. Cadmium is transported largely in the red blood cells. Selective accumulation of cadmium in the renal cortex is reported, although in cases of excessive exposure the liver may contain more. In inhalation exposure, most cadmium salts have short-term retention in the lungs. Under normal conditions, the inhalation route does not contribute significantly to the overall cadmium intake.

Acute inhalation exposure to cadmium may result in pulmonary edema and death caused by anoxia. Other acute effects include cellualr proliferation in the alveoli and hyperplasia, occasional intra-alveolar hemorrhage, peribronchial fibrosis, and emphysema.

Chronic inhalation exposure to cadmium may result in proteinuria and emphysema. Reports exist of increased incidence of tumors in animals following oral exposure to cadmium, but these are of questionable adequacy.

Cadmium is a known animal teratogen and reproductive toxicant. It has been shown to cause renal dysfunction both in humans and animals. Other toxic effects attributed to cadmium include immunosuppression (animal data), anemia (human data), pulmonary disease, bone damage, and sensory dysfunction (human data).

The reference dose for chronic oral exposure (RfD) for cadmium is 5E-4 mg/kg-day (water) and 1E-3 mg/kg-day (food) (U.S. EPA, 1985). These estimates were made on the basis that a concentration of 200 μg cadmium (Cd)/gm wet human renal cortex is the highest renal level not associated with significant proteinuria (IRIS, 1993; U.S. EPA, 1985). On the basis of a toxicokinetic model (U.S. EPA, 1985), which assumes 0.01 percent elimination of cadmium body burden per day and 2.5 percent absorption of cadmium from food or 5 percent from water, NOAEL for chronic cadmium exposure is 0.005 and 0.01 mg kg/day from water and food, respectively.

Uncertainty and modifying factors for the oral RfD are estimated at 10 and 1, respectively. The uncertainty factor accounts for intrahuman variability to cadmium toxicity in the absence of specific data on sensitive individuals. The oral RfD is based on the highest level of cadmium in the human renal cortex (i.e., the critical level) not associated with significant proteinuria (i.e., the critical effect). A toxicokinetic model has been used to determine the highest level of exposure associated with the lack of a critical effect.

The choice of NOAEL does not reflect the information from any single study but reflects the data obtained from many studies on the toxicity of cadmium both in humans and animals. These

data also permit calculation of pharmacokinetic parameters of cadmium absorption, distribution, metabolism, and elimination. All of this information considered together leads to a high confidence in the data base. High confidence in the RfDs follows.

Estimation of an RfC for chronic inhalation exposure to cadmium is currently under review by an EPA work group (IRIS, 1993).

Evidence of Carcinogenicity

Cadmium is classified as a B1 carcinogen by the EPA, meaning it is a probable human carcinogen (IRIS, 1993). This is based on limited evidence from epidemiologic studies and sufficient evidence of carcinogenicity in rats and mice by inhalation, intramuscular and subcutaneous injection.

Human Carcinogenicity Data: Data is limited. A two-fold excess risk of lung cancer was observed in cadmium smelter workers. The cohort consisted of 602 white males who had been employed in production work a minimum of 6 months during the years 1940-1969. The population was followed to the end of 1978. Urine cadmium data available for 261 workers employed after 1960 suggested a highly exposed population. The authors were able to ascertain that the increased lung cancer risk was probably not due to the presence of arsenic or to smoking (Thun et al., 1985). Because of the involvement of several confounding factors, this study offers limited evidence on human carcinogenicity. Similarly, other studies on excess lung cancer risks have not accounted for the possible exposure to other carcinogens, such as arsenic and smoking, leaving considerable limitations on the overall observations (Varner, 1983; Sorahan and Waterhouse, 1983; Armstrong and Kazantzis, 1983).

Animal Carcinogenicity Data: Studies with Wistar rats have revealed that exposure via inhalation of cadmium chloride at concentrations of 12.5, 25, and 50 $\mu g/m^3$ for 18 months, with an additional 13-month observation period, resulted in significant increases in lung tumors (Takenaka et al., 1983). Intratracheal instillation of cadmium oxide did not produce lung tumors in Fisher 344 rats but rather mammary tumors in females and tumors at multiple sites in males (Sanders and Mahaffey, 1984). Injection site tumors and distant site tumors (for example, testicular) have been reported by a number of authors as a consequence of intramuscular or subcutaneous administration of cadmium metal and chloride, sulfate, and oxide and sulfide compounds of cadmium to rats and mice (U.S. EPA, 1985). Seven studies in rats and mice where cadmium salts (acetate, sulfate, chloride) were administered orally have shown no evidence of a carcinogenic response. Results of mutagenicity tests in bacteria and yeast are inconclusive. Conflicting results have been reported on cadmium-induced chromosomal aberration, mutation, and cell transformation tests.

Carcinogenicity Risk Assessment

At present, a quantitative estimate of carcinogenic risk from oral exposure is not available. There are no positive studies of orally ingested cadmium suitable for quantitation.

A summary of the risk estimates from inhalation exposure is provided as follows:

Inhalation Slope Factor: 6.1E+0 per mg/kg-day

Inhalation Unit Risk: 1.8E-3 per $\mu g/m^3$

(Unit Risk should not be used if the air concentration exceeds 6 $\mu g/m^3$ since above this concentration the unit risk may not be appropriate).

Air Concentrations at Specified Risk Levels

Risk Level

1E-4
1E-5
1E-6

Concentration

6E-2 $\mu g/m^3$
6E-3 $\mu g/m^3$
6E-4 $\mu g/m^3$

The confidence in the carcinogenicity data is rated moderate. The data was derived from a relatively large cohort. Effects of arsenic and smoking were accounted for in the quantitative analysis for cadmium effects.

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RADIUM

Isotopes Ra-223,224,226,228

Introduction

There are three isotope decay series which give rise to the four radium isotopes described. The Thorium decay series gives rise to Ra-228,224, half-lives of 5.7 years and 3.6 days, respectively. Uranium-235 decays into Ra-223, half-life of 11.4 days. Uranium-238 decays into Ra-226, the precursor of Rn-222, and has a half-life of 1600 years. All of the above isotopes degrade at their isotope- specific decay rates, and eventually turn into stable lead. Ra-223, 224, and 226 are alpha emitters, and Ra-228 is primarily a beta emitter.

Environmental Fate and Transport

Radium is a naturally-occurring metal in the earth's crust which is part of the decay chains of Uranium and Thorium. Radium isotopes are present in the wastes of uranium and phosphate mining and refining processes. Fallout of radium is also associated with coal combustion.

Radium forms compounds with other matter which alters its stability, solubility and bioavailability. $RaBr_2$ and $RaCl_2$, for instance, are soluble in water and alcohol. $RaCO_3$ is insoluble in water and like elemental radium, decomposes in acids. Elemental radium also decomposes in water. With a valence state of +2, radium gains its toxicological significance as a calcium analoge.

The environmental fate of radium depends on its chemical form, but it is generally not a mobile constituent in the environment. Radium in water may be readily adsorbed by soils and sediments, with decreasing sorption of Ra^{2+} at higher pHs and total dissolved solids contents. It may be bioconcentrated and bioaccumulated by plants and animals and is transferred in food chains from lower trophic levels to humans. Transfer of radium from soil to plants can be described by concentrations ratios[1]. Further modelling of the radium concentration in the milk and flesh of grazing animals from forage material can be estimated with transfer coefficients as well (Watson, et al., 1984). Radium fallout from coal fly ash has been shown to concentrate in snow near power plants in Poland (Jaworowski et al. 1971).

Toxicity

Most of our data on the human health effects of radium isotopes comes from studies of radium dial painters and patients injected with Ra-224. There are also case histories of people exposed to radium, including people who have died following chronic exposures (Gettler and Norris, 1933, Martland, 1931, and Reitter and Martland, 1926). Approximately 80 to 85% of radium body burden is found in bone, with the remainder distributed uniformly in the soft tissues (United Nations, 1972), although "hot spots" are known to exist (Hoeker and Roofe, 1949 and

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[1]. A concentration ratio describes the partitioning of a compound from the host soil to the plant mass, also called the soil-plant transfer coefficient.

1951). The root cause of damage to living tissue from radium exposure is considered to be the high linear energy transfer (LET) alpha particles (BEIR IV).

Hematopoetic tissue diseases are associated with radium exposure. Chronic myeloid leukemia, panmyelophthisis, and anemia have been reported from repeated injections of Ra-224 in humans. The connection between leukemia and radium exposure has been observed but the statistical significance of these cases above expected incidence rates is not clear.

Immunological effects may be of concern to persons who are chronically exposed to radium. Leukopenia, a disease which is characterized by a decrease in the number of white blood cells in the blood, has been observed in experimental mice and in radium dial painters.

An increased incidence of cataracts has been reported in jeuveniles injected with doses of 28 μCi/kg of Ra-224, but not at lower doses.

Carcinogenic effects ofradium exposure are well documented. Due to Radium's competition with calcium, it is a bone seeker. Bone cancer is the most common consequence of radium exposure, reported in all species tested. Carcinomas of the paranasal sinuses and mastoid air cells are associated with radium exposure (Martland, 1939), resulting in hearing loss, vision problems, pain, nasal discharge, and cranial nerve palsy. Breast cancer, lung, liver and kidney cancers are also observed in conjunction with radium exposure (Spiess, et al., 1989).

There is reason to suspect that radium exposure may lead to developmental defects. Radium crosses the placental barrier and enters fetal circulation (Martland and Martland, 1950). Data from the radium dial painters is not conclusive as to radium toxicity in offspring. Radium is considered to be a heavy element which has low tendency for fetal accumulation (BEIR IV). Sufficient data on the embryotoxic effects of radium are lacking.

Children are the most sensitive population to radium exposure, since it is stored in the bone and results in a longer exposure duration. Tooth breakage, reduced bone growth, cataracts and breast tumors are all possible outcomes of radium exposure to children.

Potential for Human Exposure

Environmental exposures to radium result from the inadvertant ingestion of contaminated soils and water, as well as the inhalation of particulates, either generated as stack and/or sourcepile emissions, or the resuspension of grounded matter. Radium in unsoftened drinking water has been shown to lead to an excess incidence of bladder and lung cancer in males and breast and lung cancer in females (Bean, et al., 1982). External exposures to radium from gamma radiation, and x-rays are also possible.

Occupational exposures to radium have historically been the result of radium dial painting, which is no longer practiced. Chemists who work with radium, thorium or uranium isotopes have been exposed to radium in case histories of radium toxicity. Dust ingestion and/or inhalation in processing operations (where radium-containing ore is crushed, graded or blasted) can also lead to radium exposure. Excess incidence of lymphomas and lung cancers have been observed

among workers who process uranium ores (Archer, 1977), although, it is difficult to separate the effects of radium in this study from other radioisotopes and from potentially confounding factors.

Regulations and Advisories

AGENCY	DESCRIPTION	VALUE	REFERENCE
EPA	Occupational Derived Air Concentration	30 pCi/m3 for Ra-226 50 pCi/m3 for Ra-228	EPA-520/1-80-020 FGR No. 11, ORP (1988)
EPA ORP	Standards for Uranium and thorium mill tailings	5 pCi/g in first 15 cm of soil, 15 pCi/g below 15 cm	40 CFR 192.12 (1978)
EPA ORP	Radiation Protection: Standards for Nuclear Power Operations. Annual radiation dose equivalent to whole body	25 mrem	40 CFR 190.10
EPA ODW	MCL Radium-226,228	5 pCi/L	EPA 1989b 40 CFR 302
FDA	Levels in bottled water, Ra-226 and Ra-228	5 pCi/L	21 CFR 103.35
EPA ORP	Groundwater Protection, Combined Ra-226 and Ra-228	5 pCi/L	40 CFR, 192.32

Cancer Slope Factors

The HEAST Tables (1992) list slope factors for the four isotopes of Radium discussed. These values assume a linear, non-threshold dose-response curve. The applicability of these values to chronic, low-level exposure with possible threshold phenomenon is appropriate for risk assessment purposes. Slope factors are listed below.

ISOTOPE	ORAL SF (risk/pCi)	INHALATION SF (risk/pCi)	EXTERNAL SF (Risk/yr per pCi/g soil)
Ra-223	6.4 E-11	3.1 E-9	2.3 E-7
Ra-224	3.8 E-11	1.2 E-9	2.3 E-8
Ra-226+D	1.2 E-10	3.0 E-9	6.0 E-6
Ra-228+D	1.0 E-10	6.9 E-10	2.9 E-6

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RADON ISOTOPES 222, 220, and 218

CAS No: 14859-67-7, 22481-48-7, and 14835-02-0

Introduction

Radon is a naturally occurring radioactive gas which is generated from decay of the Uranium-238 and Thorium-232 series. There are 3 isotopes of radon; Rn-222 (Radon), Rn-220 (Thoron), and Rn-218 (Actinium). Each isotope is a colorless, odorless, tasteless, radioactive gas characterized by alpha emissions and a short half life (3.8 days, 54 seconds, and less than 1 second, respectively). Radon is an inert gas at temperatures above -61.8 °C (Bier IV, NRC 1988).

Environmental Fate and Transport

Because the half-lives of the latter two isotopes are less than one minute, radon-222 is the main isotope of concern relevant to human toxicity. Accordingly, radon-222 is the primary isotope discussed in this and further sections, and will be referred to simply as radon.

Radon is a direct decay product of Radium-226 in the Uranium-238 decay series. Radon leaves rocks and soil in a gaseous state and enters the surrounding air and water. The natural decay of radon generates alpha and gamma radiation as well as daughter products which eventually decay into stable lead. There are no natural environmental sinks for radon, although a small amount is ultimately released to the stratosphere. Limited information indicates that plants absorb both radium-226 and radon from soil, and that these compounds are subsequently bioaccumulated in plant tissue. The rate of radon emission from plants has been estimated to be twice that of the soil from which the plant was growing (Pearson, 1967). In addition, the progeny of radon are metals which show an increased propensity for soil binding, and in the case of tobacco leaves, a propensity for binding to foliage (BEIR V).

Secular equilibrium is the assumption that all isotopes in each radioactive decay series are present in equal amounts (as measured by their radioactivity). This indicates that the levels of ambient radon isotopes remain constant due to the long half-life (and therefore prevalence) of uranium and thorium, its parent products.

Mechanical flow of air and ground water in soil and a process called "alpha recoil" are the primary mechanisms of environmental transport of radon. Alpha recoil is the phenomenon where alpha particles emanating from radon, recoil in the opposite direction of initial ejection (ATSDR, 1989). Once emitted, meteorological factors such as precipitation, atmospheric pressure, and wind dictate the additional movement of radon. The formation of radon progeny as condensed nuclei encourages its adherence to particulate matter in the air. The unattached fraction is the radon which does not attach itself to aerosols immediately following formation. Soil porosity and geology can influence the ambient level of radon as well.

General Toxicity and Metabolism

Chemical toxicity from exposure to radon is primarily associated with pulmonary pathologies, such as tuberculosis (Waxweiler, 1981), emphysema, and pulmonary fibrosis (Lundin, 1971). Adverse respiratory effects have been observed in human populations in occupational settings. The main uncertainty associated with scientific epidemiological evaluation of toxic effects of radon is that confounding factors such as coal dust also contribute to toxic endpoints. It is difficult to elucidate the specific effects of radon exposure in these and other cases.

Respiratory toxicity has been documented in animal studies following exposure to radon and radon daughters. Toxic endpoints include pneumonitis, pneumonia, and pulmonary fibrosis of alveolar cells. In addition, general inflammation of the trachea, inflammation of the mucous glands, and destruction of tracheal cells has been observed.

Hematological effects have been observed in mice following acute and chronic exposure to radon. These have included decreases in erythrocytes, reticulocytes, platelets, and white blood cells (Morken, 1961).

Other systemic effects documented include significant decrease in body weight in hamsters. In addition, epidemiological evidence seems to suggest increased incidence of genotoxic effects such as chromosomal aberrations (Leonard, 1981), stimulation of DNA-repair mechanisms (Tuschl), and increase in sister chromatid exchange (Poncy, 1980).

Evidence of Carcinogenicity

Underground mining was the first occupation associated with radon and lung cancer. Uranium ores contain high concentrations of radium and radon.
Studies involving uranium and iron ore miners have consistently demonstrated that the frequency of respiratory cancer mortality increased with radon exposure (ATSDR, 1989). It is difficult, however, to separate carcinogenic effects of radon, radon-daughters, radium, and uranium, given that exposures to these compounds are usually concurrent.

Documentation that radon and its progeny can accumulate in homes has led to increased concern about domestic radon exposure. Investigations involving residential populations have documented an increase in lung cancer incidence with radon levels as low as 1.4 pCi/L of air. Because of differences in exposure parameters between miners and residential cohorts, the dosimetry and subsequent human health effects must be examined separately for these two exposed populations.

Factors influencing radon dosimetry include the physical characteristics of inhaled air (including the attached fraction), breathing patterns, and biological pulmonary characteristics (NRC, 1988). The dose of inhaled radon increases as the unattached fraction increases[1], due to the efficient absorption of the unattached radon and/or radon progeny in the airways. Accordingly, the

[1]. The unattached fraction, as described above, is the radon which does not attach itself to aerosols or suspended particles in the air.

particle size distribution of the ambient air is a major determining factor in radon dosimetry. The attached fraction of radon which adheres to dust particles can attach to minute components of the pulmonary system as well, where radon decays to its progeny, causing deleterious effects through the emission of alpha particles.

In addition, several studies have documented increased incidence of lung cancer in laboratory animals. These studies demonstrate that development of pulmonary cancer depends on variables such as exposure frequency and exposure to other pollutants (NRC, 1988).

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URANIUM

CAS No: 7440-61-1

Introduction

There are 3 naturally-occurring isotopes of uranium; U-238, U-235, and U-234. Uranium is a solid silvery metal which forms complexes with oxygen and fluoride to form various solids ranging in color from brown-black (UO_2) to yellow-red (UO_3) and green (U_3O_8 and UF_4). Soluble compounds of uranium include Uranium Hexafluoride, Uranyl Fluoride, Uranyl Acetate, and Uranyl Nitrate. By weight, it is generally assumed that >99 percent of the total naturally occurring uranium is of the isotope U-238. By activity as measured in picocuries (pCi), however, roughly 47.5 percent of total naturally occurring uranium is U-238. Another 47.5 percent is U-234, and the remaining 5 percent activity is that of U-235.

The average concentration of total uranium in soils is approximately 1.2 pCi/g, although this concentration differs with the bedrock from which the soil is formed (NCRP, 1984).

Environmental Fate and Transport

Natural processes of uranium distribution include volcanic eruption and windblown soils. Anthropogenic sources of environmental uranium include uranium mining, milling, handling, and processing, production of phosphate fertilizers and elemental phosphorous from ores which contain uranium, and improper disposal of uranium-containing wastes.

Particulate uranium is removed from the atmosphere by deposition to surface soil, plants and surface water. Resuspension of particulates from soil and plants into the atmosphere occurs. The ultimate fate of atmospheric uranium is most likely transport from surface water into sediments.

The mobility of uranium in water and soil is greatly influenced by its ionic state as well as the complexes it may form with other compounds. A valence state of +6 makes it easier for uranium to form soluble complexes than +4. Generally, the formation of soluble uranium compounds at pH's of less than 6, in waters of high organic content, makes uranium more mobile in the environment.

Uranium is not known to bioaccumulate in aquatic organisms. Fruits and vegetables grown in contaminated areas may have elevated levels of uranium as a result of windblown particles adhering to their surfaces. Generally, it has been observed that the root portion and not the above-ground portion of plants which grow in uranium-rich soils absorbs significant quantities of uranium from soil.

Increased uranium concentrations in cattle grazed in contaminated areas have been observed (Smith and Black, 1975).

Noncarcinogenic Toxicity

Chemical toxicity from uranium is primarily the result of acute renal damage and kidney failure. This is the result of soluble uranyl ion which is generated from the uranium bicarbonate complex following filtration and reabsorption, and damages the proximal tubules. Recovery can occur in less severe cases.

Inhalation exposures to uranium can lead to irritation of the lungs and nasal passages. Insoluble uranium salts are retained by the lung. Uranium tetrafluoride and uranyl fluoride have a tendency to form hydrogen fluoride in body tissues, also irritating the eyes and skin.

An oral reference dose for uranium (soluble salts) of 0.003 mg/kg-day is established by the EPA, based on a single study of uranyl nitrate hexahydrate orally administered to rabbits over 30 days (Maynard and Hodge, 1949). Confidence in this study is high, although the reference dose is given medium confidence since it is derived from an animal-based LOAEL which did not evaluate sensitive individuals. The critical effect observed is nephrotoxicity.

Although uranium crosses the fetal barrier, it is considered to be a heavy element with low tendency for accumulation in fetal tissues. Limited data for the developmental effects of uranium suggest that the main mechanism of toxicity to the fetus is the result of chemical toxicity. This is based on one study by Sikov and Rommereim (1986) which injected U-233 intravenously into pregnant rats and observed a dose-related trend in fetal malformations. Longer-term studies in both animals and humans are needed using appropriate isotopes, ie., U-235 and U-238, to address the developmental effects of uranium exposure.

Carcinogenic Toxicity

Uranium is listed as a Class A carcinogen. Slope factors have been published in the Health Effects Assessment Tables (HEAST) for assessing risk from exposure to U-238, U-235 and U-234. U-238 and U-235 are the parent isotopes for separate decay chains. The most probable effect of exposure to uranium would be an increase in bone sarcomas (BEIR IV).

Risk from exposure to the decay products of these isotopes can be adressed in part by using the "plus daughter" slope factors for U-238 and U-235, as indicated in the HEAST. Slope factors are given for external exposure (primarily gamma radiation), oral exposure, and inhalation exposures (alpha and gamma radiation). Other components of risk from uranium exposure include the risk from the *progeny* of uranium, specifically, radium-226 and radon-222.

Exposure Populations

Occupational exposures to uranium include those people involved in the mining or milling of uranium or phosphate ores, people who package yellowcake or handle enriched uranium, and people who work with uranium wastes. General populations who are environmentally exposed to uranium include people who live in areas where phosphate slag has been used for construction

materials and/or road maintenance (Melville, 1981), as well people who live in areas which have been impacted by the uranium or phosphate industries. Exposure to uranium from ingestion of root vegetables grown in contaminated soils is another route of exposure for the general public. The EPA estimates the average daily exposure to total uranium from dietary sources is 1-1.4 μg/day, 38% of which is estimated to be from the consumption of root vegetables (EPA 1985).

Regulations and Guidelines Applicable to Uranium

TLV for total Uranium (ACGIH 1990)	0.2 mg/m^3
OSHA PEL for Uranium, insoluble compounds	0.2 mg/m^3
soluble compounds	0.05 mg/m^3
NIOSH TWA for a 10-hour workshift,	
insoluble compounds	250 μg/m^3
soluble compounds	50 μg/m^3
EPA MCLG [Proposed] (IRIS)	0 pCi/l
EPA MCL [Proposed] (IRIS)	20 mg/l
EPA Annual Dose Equivalent for whole body dose	25 mrem
Reportable Quantity for Accidental Release (IRIS)	0.1 Curie (U-238) 0.1 Curie (U-235) 0.1 Curie (U-234)
Oral Reference Dose, Total Uranium (Soluble Salts) (IRIS)	3E-3 mg/kg-day

		Oral risk/pCi	External risk/yr/pCi	Inhalation risk/pCi
Cancer Risk Slope Factors (HEAST, 1992)	U-234	1.6E-11	3.0E-11	2.6E-8
	U-235	1.6E-11	2.4E-7	2.5E-8
	U-238	2.8E-11	3.6E-8	5.2E-8

TLV = Threshold Limit Value
ACGIH = American Conference of Industrial Hygenists
OSHA = Occupational Safety and Health Administration
PEL = Permissible Exposure Limit

NIOSH = National Institute for Occupational Safety and Health
TWA = Time Weighted Average
MCLG = Maximum Contaminant Level Goal
MCL = Maximum Contaminant Level
IRIS = Integrated Risk Information System

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Appendix E

Quantification of Risks from Gamma Irradiation

INTRODUCTION

As part of the Remedial Investigation (RI) at the Monsanto Site, gamma survey measurements were taken during soil and source pile sampling. Background gamma measurements were also taken at background soil sample locations. In Section 2, a preliminary screening of gamma levels was conducted. This screening process used an Upper Tolerance Limit (95/95 UTL) as the background screening criteria to compare against Site gamma readings. Within the facility boundary, gamma levels were elevated. The results of the screening indicated that outside of the facility boundaries, gamma radiation was not elevated relative to the background UTL levels.

In this appendix, measured gamma radiation was used to estimate carcinogenic risk. These risk calculations require a different approach than the traditional slope factor/reference dose method used in the main text of this document. The objective of this exercise is to provide corollary information in the characterization of risks, presented in Section 5.

The slope factor approach includes several assumptions regarding gamma exposures. For external exposure estimates, the slope factor approach models gamma irradiation. Assumptions of the model include a uniform distribution of radionuclides in the soil, and an effective areal extent and depth of contamination, such that a uniform, infinite plane of gamma radiation is likely.

For receptor locations outside of the Plant boundary, these assumption may become less valid. Information presented in the RI and the conceptual Site model suggests that aerial deposition of particulates is responsible for the irregular surficial distributions of radionuclides. This non-uniform distribution raises uncertainty in the external exposure assumptions regarding uniformity and infinite plane geometry. Thus, risks from external exposure to radionuclides based on the slope factor approach may be overestimated. By measuring gamma radiation directly, the assumptions of the slope factor methodology may be examined.

Another utility of presenting gamma irradiation risks is for comparison to benchmark dose limits (expressed in mrem/year) established by the NRC, DOE, or other EPA regulated industries. This information may also provide linkage to the Idaho Radionuclide Study (EPA, 1990) and the proposed Slag Study.

DATA QUALITY

The measured gamma readings may have a level of precision appropriate for making order of magnitude estimates of risk; however, the error (and hence uncertainty) of these readings is within the precision and accuracy of measuring risk directly from specific radionuclides (slope factor approach).

However, uncertainty is associated with the gamma radiation measurements obtained during the remedial investigation. The Geiger-Muller detector that was used to measure dose rates does not respond in a tissue-equivalent manner under low-dose environmental conditions, insofar as the instrument over-responds. Consequently, the measured gamma dose rates yielded a conservative estimate of the cancer risks associated with external exposure.

METHODOLOGY

Risk calculations presented in Appendix B suggest that external exposure to radium-226 drives the majority of risks, especially in the industrial scenarios. Because the external slope factor for radium-226 is based on external gamma exposure, the elevations of gamma readings within the facility boundaries can be compared to risks derived from activity levels (pCi/g) of Ra-226. This appendix utilizes information from the risk assessment methodology by EPA for the National Emissions Standards for Hazardous Air Pollutants (NESHAPS) for radionuclides (USEPA 1989b). The basic paradigm of the NESHAPS approach is focused upon a calculated value of 6.23E-7 risk/mRem (USEPA 1989b, Table 6-7).

By using the gamma survey data (expressed in μR/hr) and the NESHAPS value of 6.23E-7 risk/mRem, Risk is calculated as follows:

$$\text{Risk} = \text{CR} \times \text{GR} \times \text{CF} \times \text{EF} \times \text{ED}$$

Where:	CR	=	Cancer Risk (6.23E-7 risk/mRem)
	GR	=	Gamma Reading (μR/hr)
	CF	=	Conversion Factor (1 μR/hr = 9.5E-04 mRem/hr)
	EF	=	Exposure Frequency (hrs/year)
	ED	=	Exposure Duration (years)

The various exposure scenarios, as well as specific exposure parameters (i.e. EF, ED) are independent of the NESHAPS methodology. Exposure scenarios and parameters examined in this exercise are identical to those presented in Section 3 of this document.

RESULTS

Current and future industrial risks are presented in Tables E-1 and E-2, respectively. Background risks were subtracted from site risks to yield incremental risks. In the current industrial scenario, incremental risks ranged from 1E-4 (at the Baghouse Dusts and Treater Dusts areas) to 7E-4 (at the top of the slag pile).

In the future industrial scenario, incremental risks ranged from 5E-4 (at the Baghouse Dusts area) to 3E-3 (at the nodules and the top of the slag pile).

Table E-1

Current Industrial Gamma Risks

Source	CR (risk/mrem)	GR (μR/hr)	CF	Shielding Factor	days/ year	hours/ day	EF (hr/year)	ED (years)	Site Risk	Background Risk[a]	Incremental Risk[b]
Underflow Solids	6.23E-07	68.1	9.5E-04	0.45	180	6	1080	25	6E-04	2E-04	4E-04
Baghouse Dusts	6.23E-07	38.1	9.5E-04	0.00	250	1.5	375	25	2E-04	1E-04	1E-04
Nodules	6.23E-07	108.5	9.5E-04	0.00	250	1.5	375	25	6E-04	1E-04	5E-04
Treater Dust	6.23E-07	77.6	9.5E-04	0.45	250	1	250	25	2E-04	4E-05	1E-04
Slag-Top	6.23E-07	107.4	9.5E-04	0.45	250	4	1000	25	9E-04	2E-04	7E-04
Slag-Perimeter	6.23E-07	48.8	9.5E-04	0.45	250	4	1000	25	4E-04	2E-04	2E-04

(a) Background gamma activity (19.8 μR/hr) at the respective exposure.
(b) Site Risk minus Background Risk

Table E-2

Future Industrial Gamma Risks

Source	CR (risk/mrem)	GR (μR/hr)	CF	EF (hr/year)	ED (years)	Site Risk	Background Risk[a]	Incremental Risk[b]
Future background	6.23E-07	19.8	9.5E-04	2000	25	--	6E-04	--
Underflow Solids	6.23E-07	68.1	9.5E-04	2000	25	2E-03	6E-04	1E-03
Baghouse Dusts	6.23E-07	38.1	9.5E-04	2000	25	1E-03	6E-04	5E-04
Nodules	6.23E-07	108.5	9.5E-04	2000	25	3E-03	6E-04	3E-03
Treater Dust	6.23E-07	77.6	9.5E-04	2000	25	2E-03	6E-04	2E-03
Slag-Top	6.23E-07	107.4	9.5E-04	2000	25	3E-03	6E-04	3E-03
Slag-Perimeter	6.23E-07	48.8	9.5E-04	2000	25	1E-03	6E-04	9E-04

(a) Background gamma activity (19.8 μR/hr) at the respective exposure.
(b) Site Risk minus Background Risk

PART II

BASELINE ECOLOGICAL RISK ASSESSMENT

PART 2
BASELINE ECOLOGICAL RISK ASSESSMENT
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TABLES

FIGURES

ACRONYMS

BCV	Bioconcentration values
CERCLA	Comprehensive Environmental Response, Compensation, and Liability Act
COC	Chemical of Concern. A chemical associated with a Site which would cause a hazard or risk, given the modelling assumptions of the assessment.
COPC	Chemical of Potential Concern. A chemical found to be associated with a Site which may potentially threaten human or environmental health.
EPA	U.S. Environmental Protection Agency (USEPA)
EPC	Exposure Point Concentration. The chemical concentration in a specific media (soil, water, air, etc.) to which a biological receptor is exposed.
ERA	Ecological Risk Assessment
HF	Hydrofluoric acid
IAEA	International Atomic Energy Agency
IDFG	Idaho Department of Fish and Game
IRIS	Integrated Risk Information System
LOAEL	Lowest-observed-adverse-effects-level
MCC	Monsanto Chemical Company
NAS	National Academy of Sciences
NOAEL	No-Observed-Adverse-Effects-Level
RI	Remedial Investigation
RI/FS	Remedial Investigation/Feasibility Study
TRV	Toxicological Reference Value
UCL	Upper Confidence Limit of the mean value of a set of a population. Normally viewed as the 95% probability that the true mean of that population would fall below that value.
USDA	U.S. Department of Agriculture
USDHW	U.S. Department of Health and Welfare
USFWS	U.S. Fish and Wildlife Service
USGS	U.S. Geological Survey
UTL	Upper Tolerance Limit. The upper 95th percent confidence limit on the 95th percentile of a data set.
WDNR	Wisconsin Department of Natural Resources
WQC	Water Quality Criteria

EXECUTIVE SUMMARY - ECOLOGICAL RISK ASSESSMENT

This document is a baseline ecological risk assessment for the Monsanto Chemical Company (MCC) Superfund Site in Soda Springs, Idaho. It has been prepared by the U.S. Environmental Protection Agency (EPA) as part of a detailed environmental impacts study of the MCC facility known as a Remedial Investigation/Feasibility Study (RI/FS). The rest of the Remedial Investigation is being performed by Monsanto in accordance with the requirements of the Superfund law, under EPA and State oversight. This assessment will eventually be incorporated into the RI/FS Report being prepared by Monsanto.

This ecological assessment is not a completely stand-alone document, although every effort has been made to provide information about assumptions, equations, methods and values used, at least by reference. The Remedial Investigation Report will include the detailed information summarized and/or referred to in this document.

Please note that this assessment is primarily written for the technical reader who is familiar with ecological risk assessment and the references cited. This executive summary is specifically written for the lay reader who may have less technical background but an interest in the findings of the report.

FINDINGS OF THE REMEDIAL INVESTIGATION SO FAR

The production of elemental phosphorus at the MCC facility creates several byproducts and wastes (mostly heavy metals and radionuclides) which have been found in the groundwater beneath the plant and in soils near the facility at levels above background. Among the constituents of interest are cadmium, fluoride and selenium in groundwater; and arsenic, cadmium, fluoride, and selenium in soils and creek sediments.

While some uncertainties remain that may warrant further investigation, the extent of contamination appears to be limited to the MCC Plant itself and the soils immediately surrounding the Plant. Windblown dust and other airborne emissions appear to be a mechanism for contaminants to leave the site. Contamination of ground water occurs underneath and nearby the facility (see Figures 1-2 and 1-3). Infiltration of contaminants from unlined ponds into the ground water appears to be a primary mechanism.

PURPOSE AND OBJECTIVES OF THIS ASSESSMENT

The purpose of this assessment is to provide an evaluation of potential risks to plants and animals from chemical releases at the MCC Plant. Specific objectives include the following:

- Evaluation of data and identification of chemicals of potential concern;
- Identification of potential plant and animal receptors, and exposure pathways;

- Analysis of exposure and potential effects to the receptors; and,
- Characterization of risks to the environment.

APPROACH

This risk assessment provides a quantitative and qualitative description of the physical, chemical, and biological aspects of the environment; identifies areas of concern based on spatial analyses of media chemistry; evaluates exposure to various receptor groups such as plants and large and small animals; and, compares toxicological effects data with predicted intake levels for selected receptors.

The assessment is primarily based on an analysis of soil, surface water, and sediments outside of the Plant boundaries. Analyte concentrations in the samples were compared to background levels to determine the magnitude of elevation above background. Several potential contaminants were identified and carried through subsequent steps of the risk assessment. Full documentation of the nature and extent of this contamination is found in various RI reports.

To focus the ecological assessment and provide meaningful information for making decisions about the need for further action to prevent exposure, "assessment endpoints" are chosen. Assessment endpoints are defined as those describing the effects that drive decision-making, such as the reduction of key populations or disruption of community structure. The generalized assessment endpoints used to evaluate potential ecosystem risks from contaminant releases at the MCC facility include:

- Impacts to critical, sensitive or unique habitats;
- Impacts to threatened, endangered or sensitive species;
- Effects on community structure and function in the localized ecosystem adjacent to the Plant.

Exposure characterization was conducted to describe the setting, identify exposure pathways, and to quantify contaminant intake exposures to organisms representing various levels of the food chain. Organisms evaluated include plants, mice and deer. The following exposure scenarios were evaluated:

- Potential exposures to threatened, endangered or sensitive species;
- Vegetation exposed to potential phytotoxic levels of chemicals in soil;
- Ingestion of potential contaminants in soils by field mice and mule deer;

- Ingestion of potential contaminants in spring water by deer; and,

- Exposure to aquatic communities of potential contaminants in sediments and surface water.

Toxicological effects data for various plants and animals found near the plant were researched in the scientific literature in order to obtain endpoints for comparison to the predicted intake levels. Chronic and acute toxicological reference values were selected based on the exposure scenarios.

The results of the exposure characterization and the toxicity assessment were integrated by comparing estimates of chemical intake (based on reasonable maximum exposures) with appropriate toxicological endpoints to determine the likelihood of substantial effects for each receptor organism. Exposure pathways resulting in exceedances of toxicological reference values were identified.

SUMMARY AND CONCLUSIONS

Based on the available information, the findings of the ecological risk assessment support the following conclusions:

- Critical, sensitive, or unique habitats have not been impacted by releases from the MCC Plant. This conclusion was made primarily in Section 2.0, when areas of contamination were determined to not contain any of these habitats. Habitats distributions were described in Section 1.0.

- Potential impacts to threatened, endangered or sensitive species are considered negligible. This conclusion was made primarily in Section 3.0 when it was determined that: food chain transfer of contaminants to toxic levels would be negligible to the boreal owl, bald eagle and peregrine falcon; and, that these species and other sensitive species do not prefer the habitat associated with contaminated media around the Plant during any important phase of their life-cycles.

- Some risks may occur to specific organisms associated with the localized ecosystem around the Plant, although the structure and function of this ecosystem does not appear to have been altered (conclusion drawn in Section 5.0). Specific conclusions regarding these potential risks include:

  - Sensitive plant species near the northern Plant boundary may be at risk from cadmium and zinc in the soils;

  - Moderately mobile organisms (as modelled by field mice) may be at risk from cadmium and selenium, mainly north of the Plant boundary;

- Chronic exposure to other elevated metals in soil in field mice and deer would result in low toxicological risk (the predicted chemical intake exposures are less than the TRVs);

- Sensitive transient organisms (as modelled by the deer) may be at risk from fluoride concentrations in the Mormon Springs complex. Domestic stock water (e.g., for cattle and horses) from the Mormon Springs complex may also pose a hazard due to fluoride;

- Sensitive aquatic organisms are likely to be at risk from selenium and cadmium in surface water and sediments, particularly in Mormon creek and the portion of Soda Creek in the vicinity of Mormon creek.

While sufficient information was provided to perform the baseline ecological risk assessment, some issues were identified that could be fully evaluated with the available information. Three in particular are currently being evaluated by EPA to determine if more data should be collected. Those areas of uncertainty are:

- the significance of elevated levels of selenium and other site-related constituents in sediments of Soda Creek;

- the significance of effects from contaminated ground water as it intersects and mixes with surface water in various portions of Soda Creek;

- the lack of selenium data in surface water and limited selenium data in soils.

Additionally, more samples have been collected in the Soda Creek drainage area to evaluate potential contamination extent and impacts. The results of this evaluation will be available in the Spring of 1995.

Based on the results of this risk assessment, it appears that there are pathways of ecological concerns that should be evaluated to determine what, if any, action should be taken to reduce or eliminate those risks.

NEXT STEPS

Once EPA provides Monsanto with this risk assessment, Monsanto will proceed to complete the Draft Remedial Investigation Report. While that is being done, EPA will be working with the State of Idaho to identify federal and state regulatory requirements associated with the chemicals of concern.

After that, EPA will evaluate the results of the ecological assessment along with the results of the human health risk assessment and the aforementioned regulatory requirements to determine appropriate remediation goals for this site. Those goals will be used by Monsanto as the company proceeds with the evaluation of potential actions to reduce risk in what is known as the Feasibility Study.

1.0 INTRODUCTION

This ecological risk assessment is part of the Remedial Investigation/Feasibility Study (RI/FS) of the Monsanto Chemical Company (MCC) Soda Springs Elemental Phosphorus Plant. Because the RI/FS is an analytical process designed to support risk management decision-making for Superfund sites, the assessment of environmental risk plays an essential role in the RI/FS.

The primary objective of this risk assessment is to evaluate actual and/or potential chemical hazards to ecological receptors that are attributable to the MCC facility in the absence of any remedial action. Full documentation of the nature and extent of contamination, as well as background characterization is presented in the various RI reports, and will not be presented in this document.

Specific objectives of this assessment include:

- Evaluation of data and identification of chemicals of potential concern;
- Identification of potential plant and animal receptors, and exposure pathways;
- Analysis of exposure and potential effects to the receptors; and,
- Characterization of risks to the environment.

Guidance documents used to prepare this risk assessment include:

- Review of Ecological Risk Assessment Methods (USEPA 1988);
- Risk Assessment Guidance for Superfund, Volume II, Environmental Evaluation Manual (USEPA 1989); and,
- Framework for Ecological Risk Assessment, Risk Assessment Forum (USEPA 1992b).

1.1 OVERVIEW

The elemental phosphorous production facility in Soda Springs, Idaho, has been operated by the Monsanto Chemical Company since the mid-1950s. Prior to Monsanto's purchase of the property in 1952, the site was used for agricultural and domestic purposes (Golder 1992). Figure 1-1 provides the location of the site.

The production of elemental phosphorous at the MCC Plant uses a thermal process which treats the phosphate ore in electric-arc furnaces. This industrial process creates several byproducts (mainly heavy metals and radionuclides) which have accumulated in various environmental media. Approximate areas of ground water and soil contamination above background are presented in Figures 1-2 and 1-3. In order to assess risks to potentially-exposed organisms and biological communities near the Plant, this baseline ecological risk assessment was undertaken.

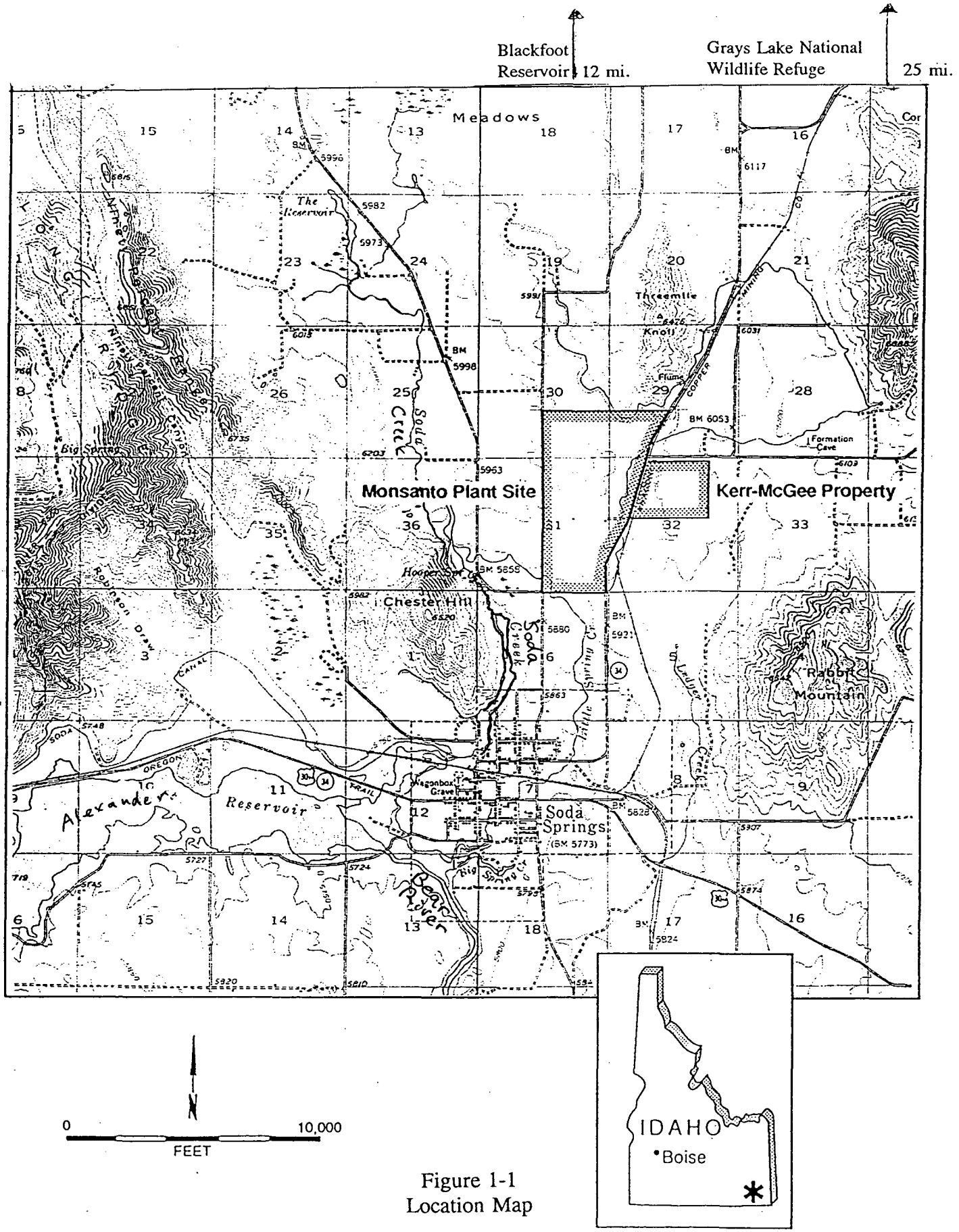

Figure 1-1
Location Map

SOURCE: Golder Associates

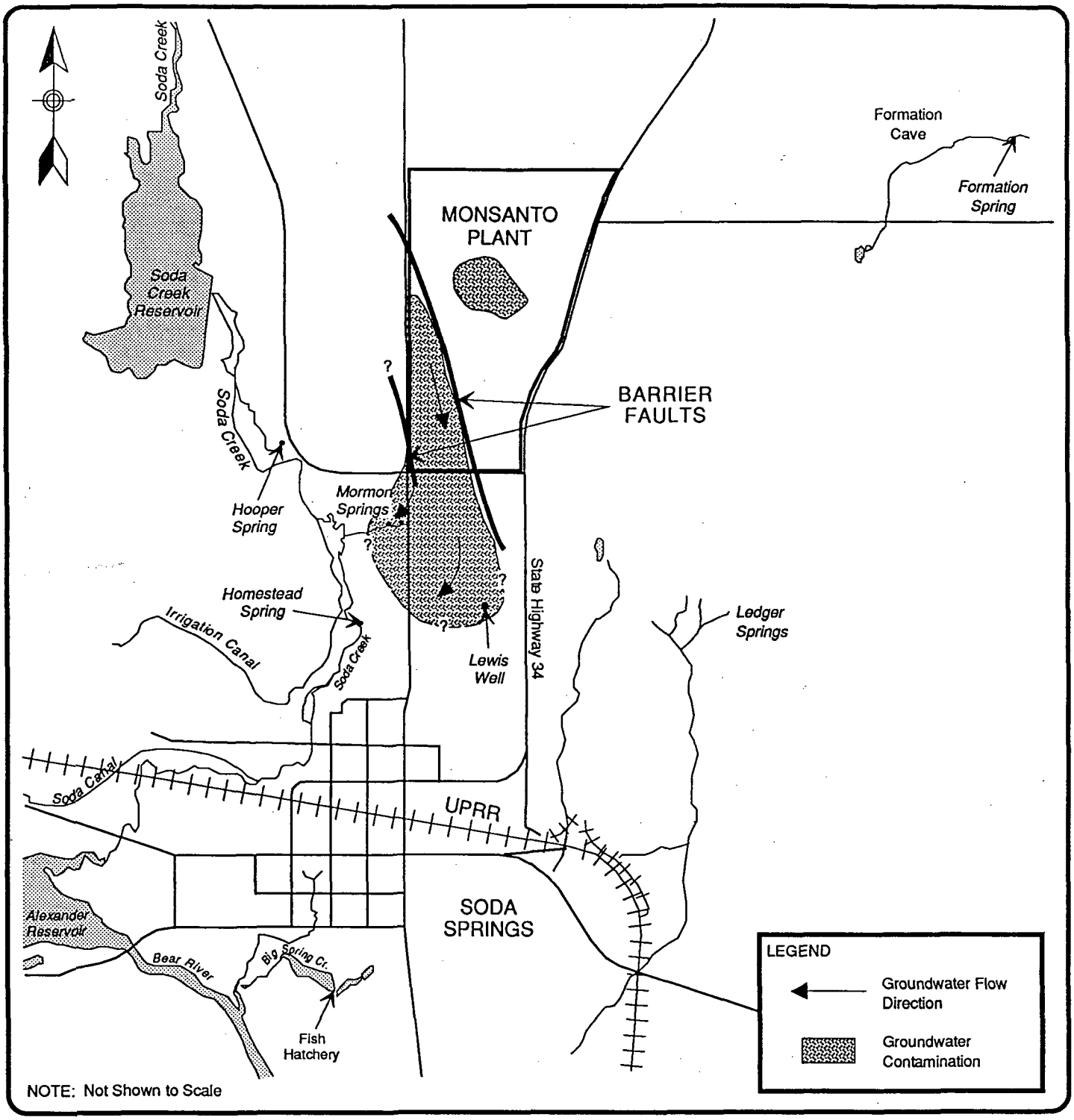

Figure 1-2

APPROXIMATE AREAS OF GROUNDWATER CONTAMINATION

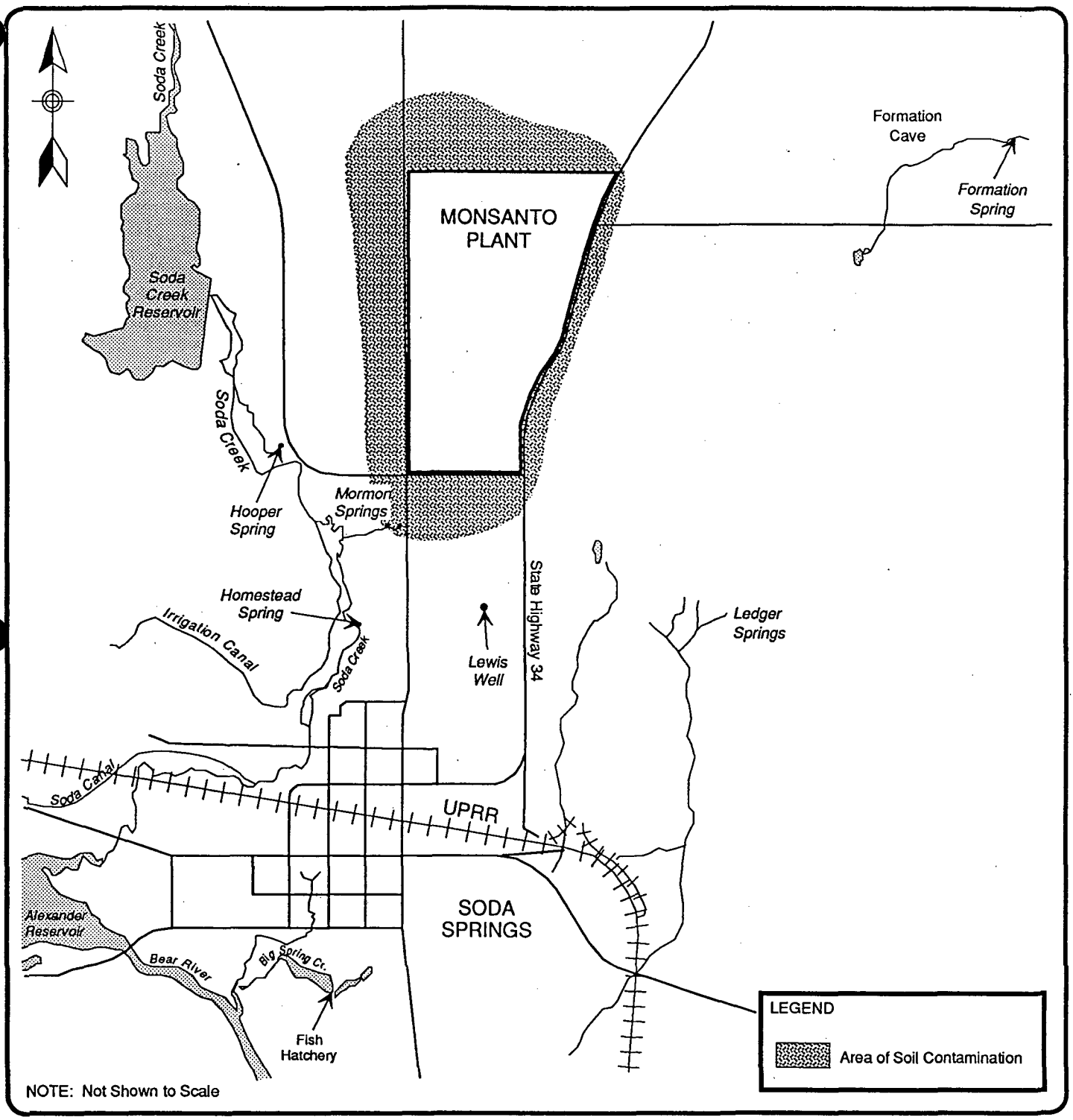

Figure 1-3

APPROXIMATE AREA OF SOIL CONTAMINATION
ABOVE BACKGROUND

The MCC Plant was placed on the Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA) National Priority List in August 1990, primarily because of potential effects on ground water quality from operational practices. Monsanto began environmental sampling to determine the nature and extent of contamination subsequent to an Administrative Order on Consent for Remedial Investigation/Feasibility Study (RI/FS) issued to Monsanto in March 1991 by EPA. Phase I data were presented in the Preliminary Site Characterization Summary Report, submitted in April 1992. Several other reports and technical memorandums regarding Phase II data have been submitted. Relevant RI data have been used in this risk assessment.

1.2 SITE DESCRIPTION

The MCC Plant is located approximately 1 mile north of Soda Springs, Idaho (Figure 1-1). Monsanto owns approximately 540 acres at this location. Portions of the Monsanto property to the south and southwest of the Plant are agricultural. Rangeland and agricultural land predominate the area immediately surrounding the Plant.

Several retention ponds exist onsite. The effluent settling pond and the sewage lagoon are unlined. The effluent pond water is used to remove solids from the non-contact cooling water. The water is then discharged into Soda Creek under a National Pollution Discharge Elimination System (NPDES) permit. The currently-used lined ponds include the phossy water and seal water ponds. Other onsite sources of potential contaminants include the following: (1) a variety of stockpiled waste materials; (2) stack emissions; and (3) fugitive dusts. A complete description of the elemental phosphorus production process is found in the Preliminary Site Characterization Summary Report (Golder 1992).

1.3 SURFACE FEATURES AND LAND USE

The Monsanto Plant is located in the Bear River Basin (Figure 1-1). The Bear River Basin is characterized by broad, flat valleys with a few scattered topographic features including cinder cones, rhyolitic domes, and uplifted fault blocks. The Plant lies at an elevation of approximately 6,000 feet in a tributary valley to the basin. Northwest trending mountain ranges, 8,000 feet in elevation, border the valley to the west. These mountains include the Chesterfield Range and the Soda Springs Hills. The Aspen Range lies approximately seven miles to the east of the Site. The northern boundary of the tributary valley is formed by the Blackfoot Reservoir, located approximately 13 miles north of the Site. Surface drainage in the valley, south of Blackfoot Reservoir, is predominantly to the south toward Alexander Reservoir.

Natural springs are important hydrologic features of the Bear River Basin. The city of Soda Springs obtains its municipal water supply from Formation Spring and Ledger Spring. Formation Spring is located hydraulically upgradient, approximately 2.5 miles east-northeast of the Monsanto Site. The Ledger Spring complex is comprised of several springs located approximately 1.5 miles southeast of the Site.

1.4 ECOLOGICAL CHARACTERISTICS

The following sections provide general information on the regional ecology of the area. This assessment will then focus on the areas of potential impact (Figures 1-2 and 1-3) which is discussed further in Sections 2.0 and 3.0.

1.4.1 Vegetation

The landscape surrounding the Plant is characterized by dry land and steppe sagebrush/grassland which occurs at the 5,000 to 7,000 foot elevation (U.S. Department of Interior, 1976). Bitterbrush, serviceberry, and snowberry are generally associated with each other, but are not codominant species. Idaho fescue, bluebunch wheatgrass, squirrel tail, sandberg bluegrass, and some indian ricegrass occur in the area. Juniper also commonly occurs. The naturally occurring species in this cover type are listed in Appendix A.

Although the agricultural cover type now occupies much of the immediate area surrounding the Plant, a great percentage of the agricultural lands were once sagebrush-grassland, riparian, and dry land. Grazing has reduced the vegetative cover, compacted the soil, and increased surface runoff and erosion in localized areas of the valley. The most common agricultural crops near the Monsanto Plant are "small grains" (wheat and barley), alfalfa hay, and pasture grass.

1.4.2 Wildlife

Significant fish and wildlife habitats near the MCC Plant include Soda Creek, the Bear River, Alexander Reservoir, and Formation Cave (a property owned by the Nature Conservancy). Gray's Lake National Wildlife Refuge is located 25 miles north of the Plant. The following sections briefly discuss the wildlife groups found in the area. See Appendix A for a list of wildlife in the region.

Game Species - The Idaho Department of Fish and Game (IDFG) identifies several big game species in this region, including mule deer, elk, and some black bear (U.S. Dept. of Interior 1976). Mule deer are the most important big game species in Idaho in terms of hunter participation and total animals harvested. The critical winter range of these species is located in the sagebrush-grass vegetation cover type upwards into the mountain brush cover type. Mule deer tend to use the same wintering grounds year after year. The MCC Plant lies within a seasonal deer migration route. Mule deer generally migrate in the winter from the Aspen Range across the valley (south of the Monsanto Plant) towards the Chesterfield Range and Blackfoot Reservoir. If migration routes are well developed, these pathways tend to be used even if they are partially blocked. Monsanto claims to periodically open Plant gates to allow deer to migrate through.

Sage grouse and sharptailed grouse exist within the sagebrush-grass vegetation type, usually near streams and bottoms of meadows. Other small game species include two species of forest grouse (blue and ruffed), two species of partridge (the Chukar and the Hungarian), snowshoe hare, white-tailed jackrabbit, cottontail rabbit, and the mourning dove.

Birds and Migratory Waterfowl - The Monsanto Plant is located within the Pacific waterfowl flyway. Over one million waterfowl migrate through southeast Idaho during the fall and spring migrations (late August through December, and late March through May, respectively). Mallard and pintail ducks make up about one-half of the waterfowl breeding population along the flyway. Waterfowl observed in the Plant area include mallard, pintail, canvasback, redhead, teals, lesser scaup, and widgeon. Canada geese are also observed throughout the migration area. Other nesting migrators in the Grays Lake area include snow geese, trumpeter swan, and whistling swan (Dames and Moore 1992).

Species inhabiting the Bear River/Alexander Reservoir area include bald eagles, white pelicans, Canada geese, in addition to several duck and shorebird species. Approximately 10 to 12 bald eagles winter in the Bear River/Alexander Reservoir area south of the City of Soda Springs (USFWS 1991).

One of the largest greater sandhill crane nesting populations in North America, concentrated mainly in the Grays Lake Refuge, occur in the area. Other birds include herons, egrets, rails, ibis, and bitterns. Also, there are numerous song and insectivorous birds throughout the valley.

Several species of raptors inhabit the area. Well-known species include the prairie falcon, various hawks, owls, osprey, golden eagles, and bald eagles.

Small Mammals and Reptiles - There are numerous species of small mammals in the Soda Springs area. Although minimal site-specific information is available regarding the natural history of this animal group, they are likely to be very important because they constitute the food base for a number of wildlife species higher in the food chain. This group includes mice, voles, ground squirrels, and muskrats. Several species of reptiles and amphibians may also be found in the plant vicinity, including the western rattlesnake, gopher snakes, and the sagebrush lizard.

Fisheries and Aquatics - Within the Bear River Drainage, several species of fish have been identified. Fish in the Bear River and Alexander Reservoir include rainbow and cutthroat trout, whitefish, suckers, dace, yellow perch, and shiners. The lower reach of Soda Creek, just above its confluence with Alexander Reservoir, also provides a marginal trout fishery for local residents. Soda Creek is the nearest natural stream to the Plant that provides an aquatic habitat. Ledger Spring and Finch Spring also provide aquatic habitat; however, Finch Spring and the upper portions of the Ledger Spring complex do not support a fisheries resource. The upper portions of Soda Creek do not support a fisheries resource due to naturally-occurring high carbon dioxide concentrations in the water. Hooper Spring is a likely source for much of the carbonation in Soda Creek.

Trout have been observed in ponds at the Nature Conservancy property near Formation Cave. However, the population resulted from stocking and may not be self-sustaining. An onsite sewage-lagoon near the southwest corner of the plant provides an attractive habitat for a variety of species. Fish, waterfowl, deer, and fox may utilize this habitat; however, because the source of the water used for the lagoon is from non-impacted wells, and is not involved in any Plant processes, it is not considered in this assessment.

1.4.3 Sensitive Species and Habitats

The U.S. Fish and Wildlife Service (USFWS 1992) has indicated that the peregrine falcon (*Falco peregrinus*) and the bald eagle (*Haliaeetus leucocephalus*) are threatened and endangered species that occur within the range of the Monsanto Plant. These two species are not considered to reside within the affected area of the Plant. Candidate species that occur within range of the Plant include the long-billed curlew (*Numenius americarius*) and the goshawk (*Accipiter gentilis*). The curlew prefers upland prairie wetlands, and the goshawk prefers forested areas. Sensitive animal species occurring within range of the Plant include the trumpeter swan (*Cygnus buccinator*) and the boreal owl (*Aegolius furereus*). These two species prefer habitats much different than the disturbed agricultural fields near the Plant.

Sensitive plant species of the area include the hoary willow (*Salix candida*) and green needlegrass (*Stipa viridula*). The hoary willow is known to occur in the Ledger Springs area. Because of extensive agricultural use of the lands adjacent to the Plant, green needlegrass is not likely to be found within the affected area. Unique ecological areas within the narrow range of the Monsanto Plant include Formation Springs Preserve (Nature Conservancy land, approximately 95 acres with unique communities of plants and invertebrates) and the adjoining "Formation Cave Area of Critical Environmental Concern" on lands administered by the U.S. Bureau of Land Management (approximately 70 acres).

The IDFG noted the Monsanto Plant's proximity to the deer migration zone. A housing subdivision north of Soda Springs has limited the width of the migration path, but the deer have continued to use it.

1.5 SCOPE AND APPROACH OF THE BASELINE ECOLOGICAL RISK ASSESSMENT

The scope of this assessment includes all potential chemical hazards to the local ecosystem that are attributable to releases at or from the MCC Plant in the absence of any remedial action. Actual and potential hazards from such releases to various ecological receptors were evaluated.

The Kerr-McGee Chemical Company operates a facility across the highway from the Monsanto site which is also a Superfund site. While that facility is beyond the scope of this assessment and separate RI/FS's are being done for each site, data from both sites were considered where appropriate. Common background soil samples were collected and used for comparison at both sites.

This assessment has been prepared by EPA using information gathered by Monsanto for the RI/FS. Risk Assessment data needs were identified in the initial planning for the RI and have been refined as additional site characterization has been done. All environmental samples collected and analyzed in the RI were evaluated for the risk assessment. Sufficient data was available to perform this assessment, although some data gaps have been identified which may need to be addressed before the RI/FS can be considered complete.

In evaluating risk from chemical stressors, it is important to maintain perspective on natural and other anthropogenic stressors. Ecological impacts from chemical stress in the environment can range from barely noticeable effects on a single organism, to greatly increased mortality to many

species within multiple trophic levels in an ecosystem. Natural disturbances such as fire, floods, or wind, as well as agricultural practices, can cause similar ranges of impacts. When evaluating risk from chemical contamination, additive or multiplicative interactions are difficult to determine because of the great complexity and likely interaction among all stressors. For this risk assessment, chemical contamination will be considered separately from other stressors.

Assessment endpoints used to evaluate potential ecosystem risks from contaminant releases at this site include:

Impacts to critical, sensitive or unique habitats - These habitats, as well as the localized habitat surrounding the MCC Plant, are evaluated by determining if chemical releases potentially impact structural, functional or emergent properties of those habitats. Chemical toxicity of media, as well as predicted or observed effects upon biological organisms are used as measurement endpoints to evaluate assessment endpoints. Impacts to critical, sensitive, or unique habitats are evaluated by comparing spatial distributions of contaminated media to spatial distributions of these habitats. If an overlap occurs, or if a potential transport mechanism is identified, then potential effects are evaluated.

Impacts to threatened, endangered or sensitive species - Threatened and endangered species are evaluated primarily by their proximity and use of potentially affected areas around the MCC Plant, and in terms of predicted effects based upon food chain modelling.

Effects upon the community structure and function in the localized ecosystem adjacent to the Plant - Effects are evaluated by evaluating media quality in the localized habitat, and by evaluating exposure and potential effects of localized receptors.

Specific measurement and assessment endpoints will be developed upon an evaluation of the data, the spatial distribution of chemicals of potential concern, and an evaluation of potential organism exposure (Sections 2 and 3).

As a conservative (protective) approach, this assessment will evaluate potential exposures and subsequent risks from the more obviously contaminated areas. If substantial potential risk is evident, less contaminated areas will also be evaluated. This approach is taken as a means to focus the risk assessment.

Underlying principles used in this risk assessment are outlined as follows:

- Ecological receptors exist in the environment as individuals, populations, communities, and ecosystems.
- Contaminants need to exist in the environment at sufficient concentrations to cause impacts to potential receptors.
- Potential receptors need to be sufficiently exposed to contaminants for an impact to occur.

The following general steps will be taken in this risk assessment: 1) Determine areas of contamination where habitat exists; 2) Determine likely receptors within the habitats; 3) Assess chemical exposure to receptors; 4) Compare predicted exposures to known toxicological effects from chemical contaminants; and, 5) Synthesize a characterization of risk based on quantitative comparisons, qualitative observations, site conceptual models, and uncertainty.

The remainder of the document is organized to follow these general steps.

Section 2.0 - Data Evaluation and Determination of Areas of Concern. The purpose of this section is to delineate those areas associated with the Site where elevated levels of chemicals may impact ecological receptors. Quantitative and qualitative information of the Site area will be assessed concurrently. Areas of potential concern will initially be identified based on levels of contaminants found at concentrations above background, historical data, information and testing conducted during the RI, and qualitative indicators. Within these areas, potential habitats are identified. These habitat delineations are carried into Section 3.0 as areas where receptors are likely to incur the greatest exposure.

Section 3.0 - Characterization of Exposure. The habitat groups from Section 2.0 are discussed in greater detail and scenarios are developed to represent the current exposure of ecological receptors to contaminants. At this point, general habitats and receptors are classified according to the assessment endpoints. Analytical data is used directly to measure exposure concentrations to non-mobile receptors, and to calculate intake values for animal species. Habitat and receptors evaluated via qualitative analysis are also discussed. Fate and transport, relating to current and potential future exposure concentrations in various media are discussed.

Section 4.0 - Characterization of Ecological Effects. The toxicological properties of site-related chemicals are discussed. Risk-based effect concentrations, derived from research studies, are used to assess potential effects upon individual organisms. Non chemical-specific effects (i.e. those simulated in biological toxicity tests) are briefly discussed. Multiple trophic level impacts are discussed qualitatively.

Section 5.0 - Risk Characterization. Information developed in Sections 3.0 and 4.0 are combined to characterize potential ecological risk from chemicals at the Site. Projected impacts are compared to reference conditions. The characterization of risk is a synthesis of information based on quantitative comparisons, qualitative observations, site conceptual models and uncertainty of these components, all analyzed in a weight-of-evidence approach. Qualitative analysis is relied upon in this weight-of-evidence approach due to large uncertainties in quantitative estimates. Induction from qualitative analysis provides an indication of potential effects; it is used as an instrument of evidence among other qualitative and quantitative indicators. This "common sense" approach is used to construct reasonable estimates of potential ecological risk.

Section 6.0 - Summary and Conclusions. The results of the ecological risk assessment at MCC are discussed.

Section 7.0 - References.

2.0 DATA EVALUATION AND DETERMINATION OF AREAS OF CONCERN

This section identifies chemicals of potential concern (COPCs) and delineates those areas associated with the site where the COPCs may impact ecological receptors. Data are summarized, and general habitat types within the potentially contaminated areas are briefly discussed. Specific exposure scenarios are discussed in Section 3.0.

For this assessment, a conservative screening process was used to identify the COPCs (chemicals exceeding their respective background concentrations in a particular medium). Maximum concentrations in environmental samples have been compared either to maximum background values or Upper Tolerance Limit (UTL) values to determine exceedance of background. When the background sample size was approximately 15-20, the UTL was used, otherwise the maximum background concentration was used.

A qualitative analysis grouped sampling locations to accommodate the following spatial ranges of receptor groups:

- highly localized - plants, soil and sediment invertebrates.
- moderately mobile - small burrowing animals, rabbits, etc.
- transient - waterfowl, deer, songbirds, etc.

These ranges represent average receptors in non-sensitive portions of their life-cycles. However, sensitive life-stages have been considered when evaluating receptor classes (e.g. a transitory waterfowl may have been considered moderately mobile if contaminated habitat provided an attractive nesting area for that receptor).

Data are summarized for each of these spatial ranges. A quantitative ecological risk evaluation will be conducted for the areas of concern; other areas will be discussed qualitatively.

2.1 SOILS

A review of the soil chemical data collected during the remedial investigation shows that elevated concentrations of some metals, radionuclides and fluoride exist in soils, within approximately two-thousand feet of the MCC Plant perimeter. The highest COPC concentrations appeared to be within five hundred feed of the north perimeter. This is expected because, during visual observations, windborn transport of particulate material was observed. Full documentation of these processes is presented in the RI.

Because only a few soil samples were analyzed for selenium, characterization of selenium is considered incomplete with the limited available data. However, selenium data from the few samples are utilized in the assessment.

Soluble fluoride data is used in this assessment. Insoluble calcium and magnesium fluoride may become more soluble in acidic conditions (e.g., the gut of an animal). Some RI data collected indicate fairly high concentrations of these two insoluble salts; however, most of the fluoride data collected during the RI process represents soluble fluoride. Soluble fluoride measurements may underestimate the bioavailable fluoride in the soil; however, fluorite (CaF_2) is known to be much less toxic than some more soluble forms (e.g., NaF). The calcium-rich soils of the Soda Springs area likely bind most fluoride as fluorite.

2.1.1 Identification of Chemicals of Potential Concern in Soil

The following is a multi-step screening process designed to identify ecological COPCs attributable to the MCC Plant:

Comparison to Background - Table 2-1 compares maximum soil concentrations (0-1" samples) to background soil concentrations (0-1" data). Maximum background concentrations, as well as UTLs calculated from the combined MCC and Kerr-McGee background data set, have been used. UTLs used in the assessment represent a 95 percent confidence level that 95 percent of background samples fall below the UTL concentration.

The UTLs are somewhat sensitive to non-normal distributions. Based on an analysis of data distribution, background concentrations were generally lognormally distributed and were *ln*-transformed before calculating the UTLs. Most maximum soil concentrations were closely approximated by their respective UTLs. Maximum site sample concentrations that exceed background have been carried forward to the next step. As noted in Section 2.0 of the Human Health Risk Assessment, gamma readings outside of the Plant boundaries did not exceed background.

Magnitude of Exceedance - An additional analysis was conducted to assess the magnitude of chemical exceedances above background soils. Chemicals with maximum site concentrations between one and two times background, and whose total frequency of background exceedance was less than 25 percent, were not considered to be significantly elevated above background. From Figure 2-1, the chemicals of potential concern identified in soil are:

2-10 x Background	10-100 x Background	>100 x Background
Arsenic	Vanadium	Selenium
Radium-226	Silver	
Uranium-238	Chromium	
Thorium-230	Lead-210	
	Cadmium	
	Polonium-210	
	Zinc	
	Fluoride	

Chemicals not considered COPCs because of low magnitude of exceedances includ aluminum, beryllium, manganese, and molybdenum.

Table 2-1
0-1" Soil Concentration Data
Offsite Soils Compared to Background Soils

Analyte	Site Sample Range (mg/kg)	Background		Carried Forward
		Maximum	UTL[d]	
Aluminum	6,560 - 30,200	18,200	19,187	Y
Arsenic	0.4 - 34	5.4	6.1	Y
Beryllium	0.8 - 4	1.7	2.7	Y
Cadmium	0.55 - 168	9.7	9.3	Y
Chromium	8.3 - 325	21.0	23.3	Y
Fluoride[a]	2.25 - 136	6.1	4.9[c]	Y
Manganese	170 - 1,380	696	807	Y
Molybdenum	ND - 2.9	1.7	1.6	Y
Selenium[a]	ND - 109.0	0.4	NC	Y
Silver	0.03 - 13	1.0	NC	Y
Vanadium	14.7 - 467	42.0	36.0	Y
Zinc	52.5 - 2,670	123.0	112.6	Y
Radionuclides (pCi/g)				
Lead-210+D	0.8 - 65	3.2	4.0	Y
Polonium-210	0.1 - 77	3.8	3.7	Y
Radium-226+D	0.4 - 17	2.7	2.9	Y
Thorium-228+D	0.4 - 1.6	1.6	1.7	N
Thorium-230	0.2 - 18	2.1	2.1	Y
Uranium-238+D	0.42 - 16	1.4	2.4	Y

a = Values based on soluble fraction.
b = Values represent very limited data set.
c = Outlier thrown out before treatment of data.
d = Log normal conversion before UTL calculations.
NC = Not Calculated due to small sample size
ND = Not Detected

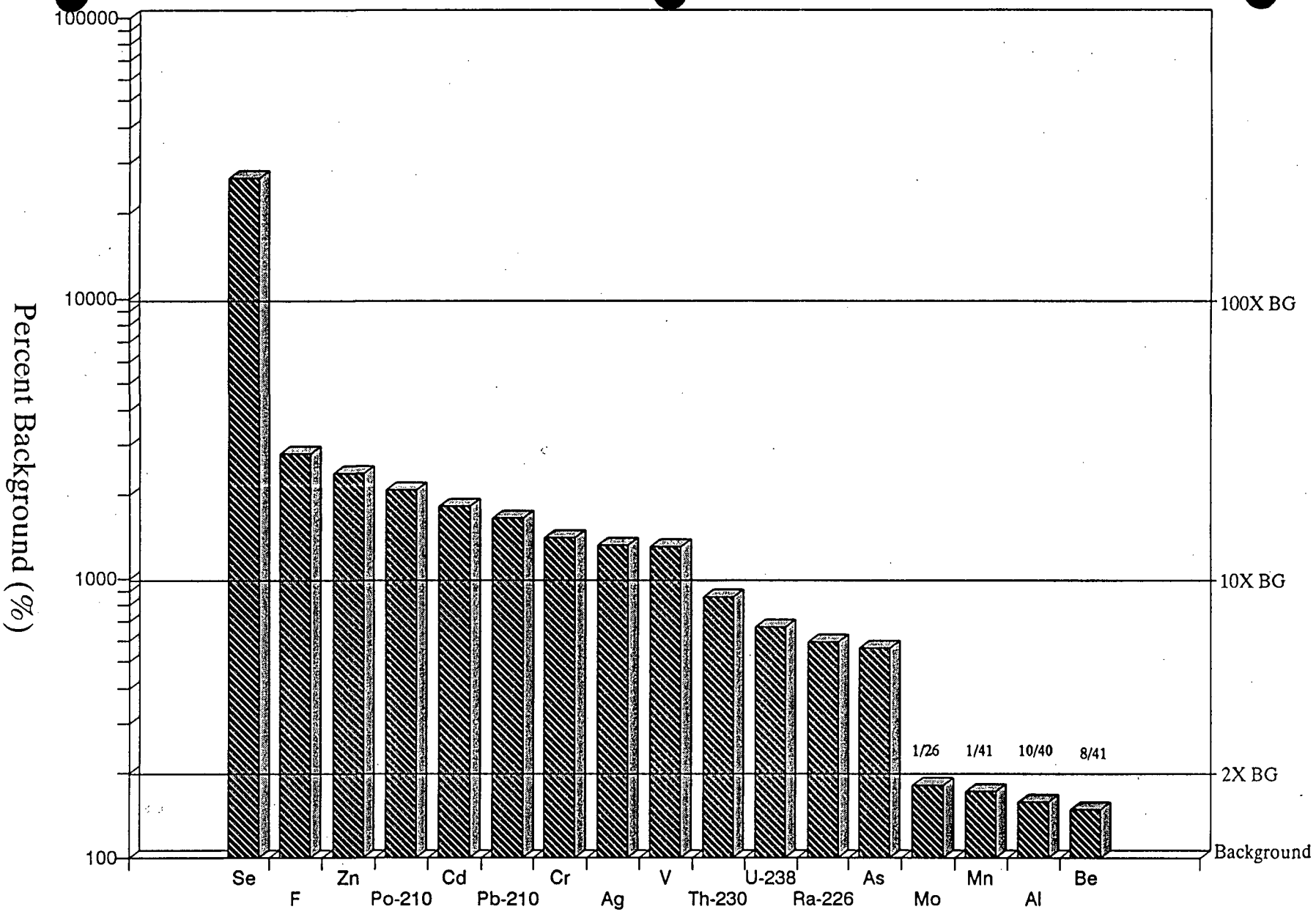

Figure 2-1

Fraction above bar indicates frequency exceeding background
UTL Concentration

Magnitude of Chemical Exceedances
Over Background

2.1.2 Spatial Analysis

A qualitative spatial analysis of the distribution of the chemicals was conducted to determine areas or locations where COPCs are most concentrated. Sampling locations are shown in Figure 2-2. Figure 2-3 shows the locations of maximum concentrations of COPCs in soils. Figure 1-3 shows the approximate location where organisms exposure to COPCs are considered. It is evident the maximum concentrations of contaminants occur most frequently at soil sampling locations S2-11 and S-13. Other sampling locations which contained maximums, as well as substantial elevations of similar contaminants, include; S2-03, S-4, S-10, S-14, and MS2-7.

Concentrations appear to be most elevated near the perimeter of the plant and may represent localized contamination linked to specific onsite sources. Contamination appears to be most highly concentrated in soils just north and northwest of the Plant. Elevated concentrations also exist, but to a lesser degree, offsite to the south. Based on the localization and general north/south partition of contamination, data are grouped into receptor groups for eventual application to exposure modelling.

Based on the distribution of COPCs in the soils, it appears that habitats considered critical, sensitive, or unique are not impacted by soil contaminants.

Three general spatial scales were assumed when delineating localized habitats. Plants and soil invertebrates occupy the most localized scale (highly localized receptors). Small animals occupy a larger (spatially) habitat scale (moderately mobile receptors), and the range of large animals generally occupies an even larger scale (transient receptors). Receptors within these spatial ranges are differentially exposed to chemical contamination based on different residence times within contaminated areas.

COPC exposure to highly localized receptor groups such as plants and soil invertebrates is best represented by single sample modelling. As a conservative approach, maximum concentrations of COPCs are used as exposure concentrations. If these concentrations pose negligible risk to the receptors, then lesser concentrations at other locations also pose negligible risk. Maximum values were found at sampling locations S2-11, S-13, S-14, and MS2-7 (Figure 2-3). These exposure concentrations are listed in Table 2-2.

Exposure to moderately mobile organisms such as voles, mice, or rabbits is best modelled by grouping data from sampling stations either on the north or south end of the Plant. Based on observations during field reconnaissance and a review of the data, two clusters of samples encapsulate habitat ranges of rodents. Clusters of sampling locations (as shown in Figure 2-3) have been constructed as follows:

Cluster NW	Cluster N
(S-10, S2-11)	(S-13, S-14, MS2-7)

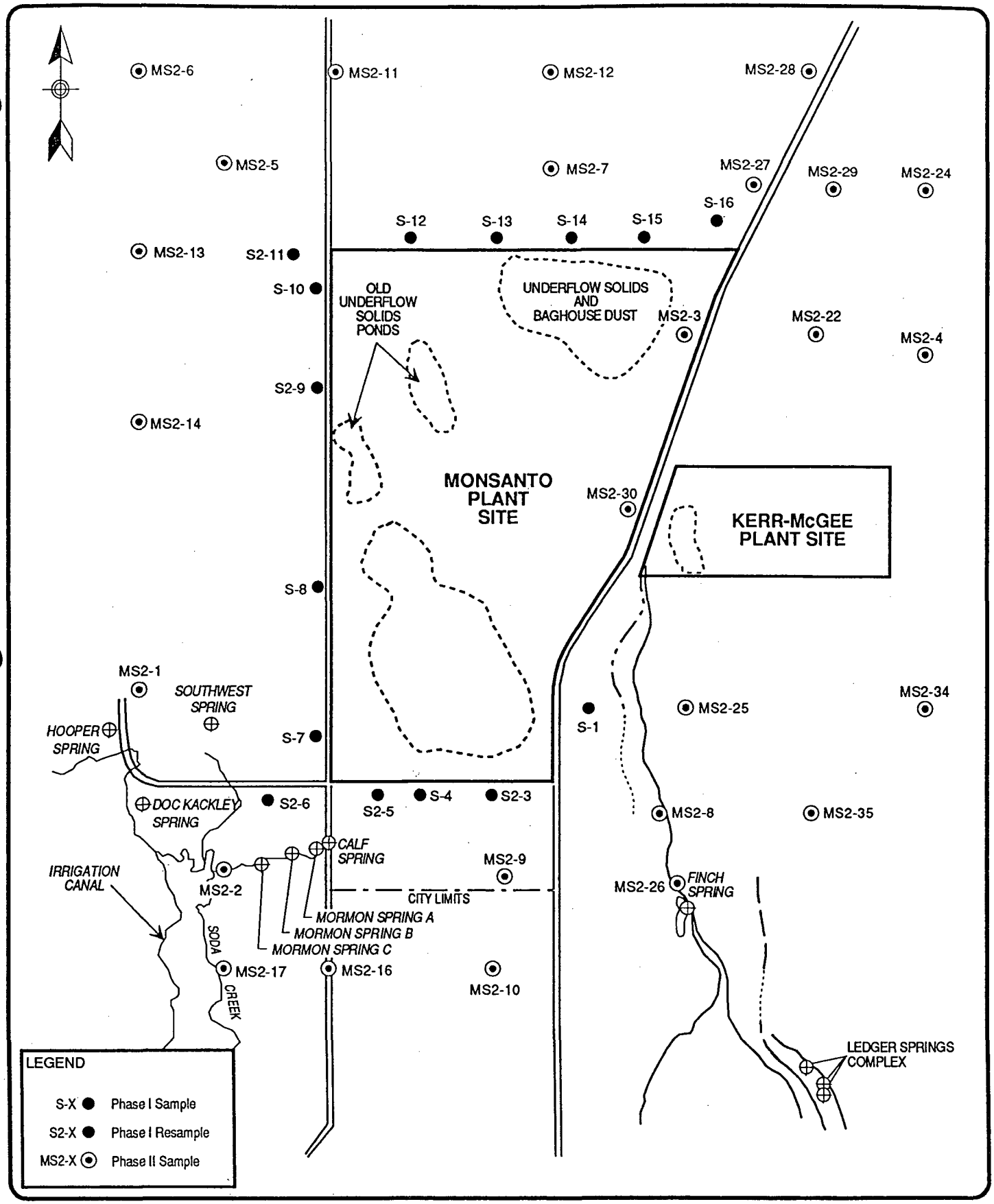

Figure 2-2

SOIL SAMPLING LOCATIONS
MONSANTO CHEMICAL COMPANY

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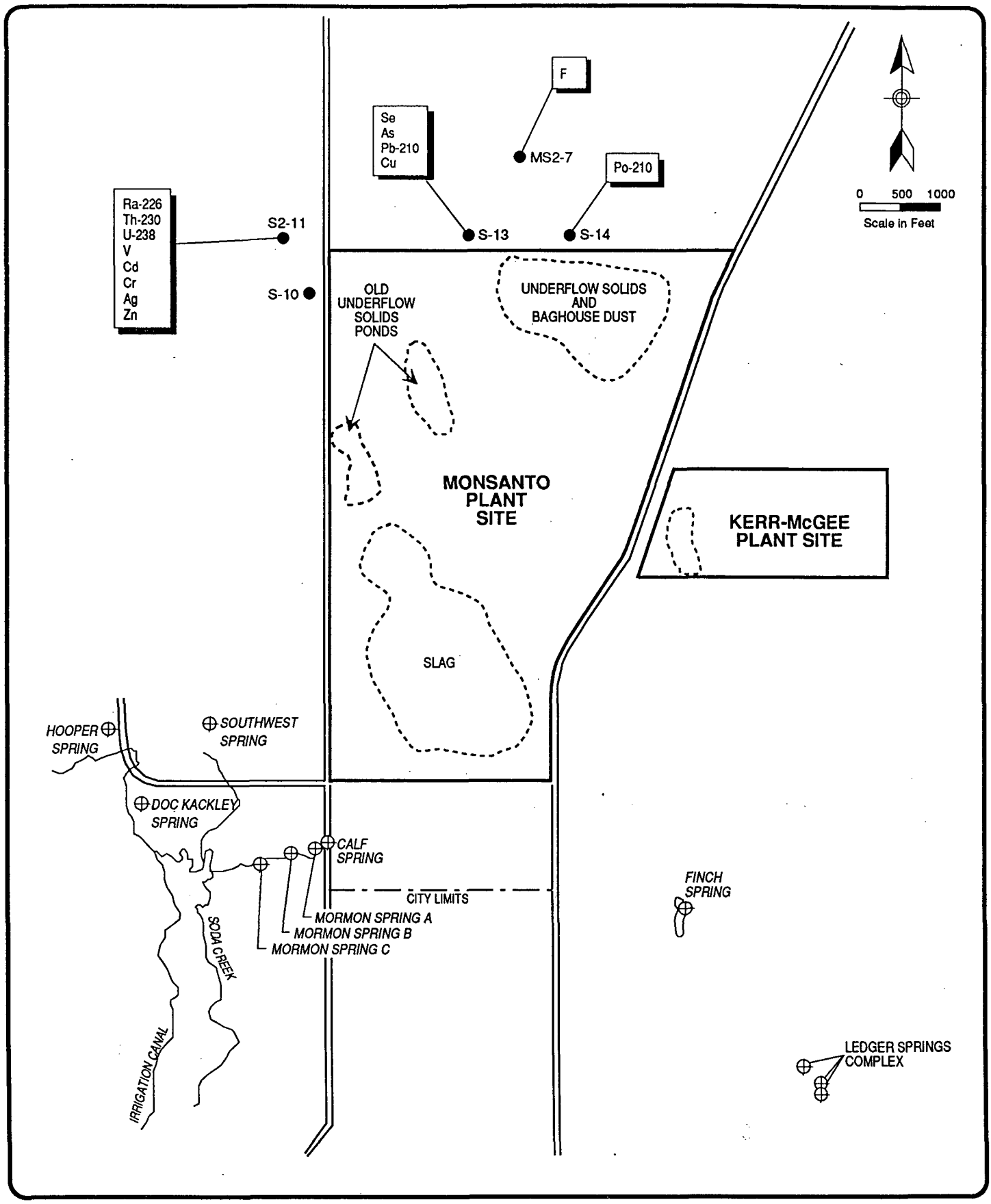

Figure 2-3

LOCATIONS OF MAXIMUM SOIL CONCENTRATIONS (0-1') OF POTENTIAL CONTAMINANTS
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(mg/kg or pCi/g)

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Table 2-2
Data Summaries for Varying Spatial Scales in Soils

Analyte	Highly Local Receptors (max conc.) n=1		Moderately Mobile Receptors (RME conc.) n=1-3		Transient Receptors (UCL conc.) n=6-9
(mg/kg)	Location	Concentration	Cluster	Concentration	Concentration
Arsenic	S-13	34	N	26.1	9.7
Cadmium	S2-11	168	NW	122.4	38.9
Chromium	S2-11	325	NW	237.5	89.5
Fluoride	MS2-7	136	N	99.7	29.5
Selenium[a]	S-13	109.0	N	59.4[b]	21.9
Silver	S2-11	13	NW	9.5	5.2
Vanadium	S2-11	467	NW	399.0	192.8
Zinc	S2-11	2670	NW	2225.0	562.1
Radionuclides (pCi/g)					
Lead-210	S-13	65	N	50.3	14.3
Polonium-210	S-14	77	N	44.3	12.6
Radium-226	S-10	17	NW	13.5	7.9
Thorium-230	S-10	18	NW	11.9	9.2
Uranium-238	S-10	16	NW	12.5	7.0

a = Selenium values represented by a very limited data set.
b = Represented by S-13II and MS2-07.
c = Represented by S-1II and S-9II.

To maintain the approach of analyzing risk for chemicals in the locations where concentrations are the highest, chemical specific data will be linked to the respective clusters where the maximum concentrations exist. For example, the maximum concentration of selenium is found at S-13 (Cluster N); therefore, selenium will be evaluated only in Cluster N. All COPCs are evaluated using this cluster method for moderately mobile receptors. As part of the qualitative review, it was assured that maximum concentrations were not isolated; that is, the adjoining samples within a cluster also had relatively high concentrations when compared to all other samples. An exception to this data clustering method occurs for selenium, which has reliable data for only a few sampling locations. Mean concentrations of COPCs within the clusters will represent the exposure point concentration (EPC) for this class of receptors. An upper bound estimate of the mean (i.e. Upper Confidence Limit) was deemed overly conservative, because selection of the most contaminated samples already biases the EPC to represent reasonable maximum exposure. If mean cluster concentrations pose negligible risks to receptors, then lower EPCs within other habitat ranges will also pose negligible risk. This is a conservative approach, because the receptors may range beyond the sample area, thus reducing exposure beyond what has been assumed. Table 2-2 shows calculated averages for the COPCs.

Transient receptors such as deer, fox, coyote, raptors, and song/game birds potentially traverse the entire perimeter of the Plant. However, deer migration is known to occur near the southern boundary of the MCC Plant. The area south and southwest of the Plant appears to provide more likely habitat to known transient receptors than other areas surrounding the Plant. This is because of the deer migration corridor, the presence of fewer plowed fields and water sources (i.e., springs and Soda Creek). Therefore, data from the southern areas have been grouped to represent exposure concentrations for transient receptors. An upper-bound estimate of the mean concentration is used to approximate reasonable maximum exposure. Concentrations of all chemicals measured are averaged from the following sampling locations: S2-3, S-4, S2-5, S2-6, S-7, MS2-1, MS2-2, and MS2-9. EPCs for this receptor group are also presented in Table 2-2.

2.2 WATER

Water resources currently or potentially affected by contamination from the Monsanto Plant include Soda Creek and several springs impacted by ground water. The springs evaluated in this assessment include Calf Spring, Mormon Springs (A, B, and C), and Southwest Spring. A review of water data reveals some elevations of metals and fluoride.

2.2.1 Identification of Chemicals of Potential Concern in Water

The forementioned spring waters have been compared to Formation Spring and Ledger Spring waters, which have been shown in RI sampling to be uncontaminated, and are assumed to be representative of background conditions. Chemicals in Mormon, Calf and Southwest Springs water are represented by multiple sampling events. Maximum concentrations in spring waters were compared to maximum background concentrations. Table 2-3 shows these chemicals that are carried forward as COPCs. As with soils, the ecologically tolerant chemical concentrations of potassium, sodium, and magnesium are not considered excessive for retention as COPCs.

Table 2-3
Chemicals Detected in Spring Water (mg/l)

Chemical	Sample Range[a]	Maximum Background Concentration	Carried Forward
Cadmium	ND - 0.018	ND	Y
Nickel	ND - 0.03	ND	Y
Selenium	0.045 - 0.19	0.011	Y
Zinc	ND - 0.151	ND	Y
Fluoride	1.1 - 3	0.29	Y
Chloride	21 - 133	7	Y
Sulfate	60 - 220	30	Y
Nitrate	2.14 - 5.2	ND	Y

a = Data from Calf Springs, Mormon A, B, C, and Southwest spring.
ND = Not Detected

Analytical results from Soda Creek water collected downstream of the effluent line (single sampling event) were compared to reference concentrations collected upstream of the effluent line (maximum concentration of three upstream control samples). Detected chemicals exceeding reference concentrations in Soda Creek are highlighted in Table 2-4 and retained as COPCs. Reliable selenium data for Soda Creek water is not available; selenium may be a COPC.

2.2.2 Spatial Analysis

Spatial analysis of the data distribution was not conducted because likely exposures would be highly localized. Maximum chemical concentrations from a pooled creek/springs data set will be compared directly to applicable water quality benchmarks. Maximum concentrations also will be used as input into transient-receptor intake models in Section 3.0. Transient receptor groups are assumed to visit these water bodies infrequently.

2.3 SEDIMENTS

A list of COPCs in Soda Creek sediments was developed during the RI process, based on conceptual site models and chemical concentrations exceeding background.

2.3.1 Identification of Chemicals of Potential Concern in Sediments

Three background samples were taken upstream of the effluent line; and sediment samples were taken downstream at various distances from the effluent line. Table 2-5 shows the screening of

Table 2-4
Chemicals Detected in Soda Creek Water (mg/l)

Surface Water	Sample Range	Maximum Background Concentration	Carried Forward
Cadmium	ND - 0.011	ND	Y
Fluoride	0.31 - 0.43	0.31	Y
Chloride	25 - 159	15	Y
Sulfate	42 - 96	36	Y
Nitrate	1.14 - 4.8	0.74	Y

Note: Reliable selenium results are not available for Soda Creek water. Selenium may be a COPC.
ND = Non-detect

Table 2-5
Chemicals Detected in Soda Creek Sediments
(mg/kg)

Sediments	Sample Range	Maximum Background Concentration	Carried Forward
Arsenic	2.8 - 49.3	8.6	Y
Cadmium	8.3 - 61	13.9	Y
Nickel	15.7 - 153	62	Y
Selenium	ND - 347	0.6	Y
Silver	ND - 1.3	0.1	Y
Vanadium	8.4 - 208	30	Y
Molybdenum	ND - 6.4	6.5	N
Copper	4.0 - 42.4	5	Y
Polonium-210 (pCi/g)	ND - 3.3	0.9	Y

ND = Non-detect

COPCs in Soda Creek sediments. Only sediment samples between 100 feet and 2,400 feet downstream of the outfall (7 samples) and the Mormon Creek sediment sample were evaluated for screening purposes. Sediment samples further downstream did not show consistent elevations of COPCs which could be attributed to the MCC facility.

2.3.2 Spatial Analysis

As with water samples, spatial analysis of the sediment chemistry data was not conducted because ecological receptors are considered highly localized. Maximum chemical concentrations from the sediment data set will be compared directly to available sediment quality benchmarks (as provided by Bennett and Culbage 1991).

2.4 DATA UNCERTAINTIES

Data uncertainty is derived from several sources. The application of quantitative and qualitative data to exposure scenarios is a primary source of uncertainty. Simplified exposure scenarios are developed based on information and observations of the site area. Varying temporal and spatial components of habitat, however, make application of "snapshot data" difficult. Fate and transport mechanisms of the chemicals also obscure the temporally and spatially fixed image of contamination. Selenium data in surface water, and to a lesser degree in soils, is also a source of increased uncertainty, due to analytical problem during Phase I of the RI. Sediment contamination downstream of the areas considered in this assessment is unclear. While there is a general decrease in sediment COPC concentrations in the lower reaches of Soda Creek, the variability in the data obscures attribution to the site.

2.5 SUMMARY OF CHEMICALS OF POTENTIAL CONCERN

Table 2-6 summarizes the list of COPCs in all media. These chemicals will be evaluated further in this risk assessment.

Given the distributional information presented in Section 1 regarding critical, sensitive and unique habitats, and the apparent distribution of COPCs associated with the Monsanto Plant, it can be concluded at this point that these habitats are not within the spatial range of contaminated areas. COPC transport to threatened or endangered species via food chain transfer will be evaluated further in Section 3.

Table 2-6
Summary of Chemicals of Potential Concern in
Soil, Spring Water, Sediments, and Soda Creek Water

COPC	Soil	Spring Water	Sediments	Soda Creek Water
Arsenic	X		X	
Cadmium	X	X	X	X
Chloride		X		X
Copper			X	
Fluoride	X	X	X	X
Molybdenum		X		
Nickel		X	X	
Nitrates		X		X
Selenium	X	X	X	
Silver	X		X	
Sulfate ion		X		X
Vanadium	X		X	
Zinc		X		
RADIONUCLIDES				
Lead-210	X			
Polonium-210	X		X	
Radium-226	X			
Thorium-230	X			
Uranium-238	X			

3.0 CHARACTERIZATION OF EXPOSURE

3.1 EXPOSURE PROFILE

The Monsanto Plant is bounded by dry land barley farming on portions of the north and west sides, with limited farming to the south. There is also onsite agricultural use in the northeast portion of the Plant Site. Much of the farming has occurred since the late 1940s. A large portion of the field north of the Plant was replanted with a grass seed mix as part of the Soil Conservation Service voluntary crop reduction program.

Southwest of the Plant, a pasture used predominantly for horses exists. Southwest Spring is located within the pasture. Further to the south is the former (b) (6) ranch, which has served as pasture for horses and cattle. During a field reconnaissance in 1991, approximately 25 head of cattle were grazing in the Mormon Creek watershed. Subsequent to that reconnaissance, MCC has stated that cattle grazing on their property near Mormon Springs will be discontinued. Fluorosis in cattle allegedly occurred prior to installation of air control technologies in the late 1980's (B. Geddes, personal communication, 1993).

The Soda Creek drainage south of Hooper Springs is grazed by horses and cattle. During the site visit, a mule deer was observed on the slope approximately 200 yards west of the creek near the Monsanto effluent discharge pipe. Downstream of the Monsanto effluent line, Soda Creek receives recharge from several springs, with most of the recharge derived from the Mormon Springs complex and Southwest Spring. It appears that aquatic habitat in Soda Creek, near the MCC Plant, is naturally harsh (due to high levels of carbon dioxide in the water), and does not support a rich ecosystem. The creek does not support a trout fisheries resource, except near the confluence with Alexander Reservoir. Other species such as carp may be found much further upstream (personal communication with Ted Norton, Golder Associates, July 1993).

The habitat conditions of Soda Creek are not considered critical, sensitive, or unique. The drainage provides minimal, if any, nesting habitat for migratory waterfowl.

The Monsanto Plant is fenced around the entire perimeter with roads bordering the site on three sides. Small mammal tracks were observed along the north property line. Habitat use by larger animals is greater in the area southwest of the Plant. A winter migration route used by deer generally follows a path from the Rabbit Mountain area (east), through the Ledger Creek/Springs complex, across Highway 34 north of the trailer court, to Soda Creek and along the Chesterfield Range.

Avian species are often attracted to the larger rangeland areas of Ledger Creek, Soda Creek, and the Three mile basin area, more so than in areas surrounding the Plant. To a limited extent, waterfowl and birds are attracted to the MCC sewage lagoons; ducks have been observed feeding in the lagoons. These lagoons contain non-process water and are not considered in the assessment as an exposure pathway. The seal water pond is not considered an attractive nuisance for waterfowl due to a lack of vegetation along the pond edges, and worker activities in the vicinity.

Because of the elevated COPCs in soil, exposure pathways of concern include plant uptake of COPCs and soil ingestion by animals. Limited food chain transfers also may occur. Another potential exposure route may be through direct foliar absorption of volatilized airborne COPCs, although this pathway appears to be minor. This pathway may have been more important in the past; however, the installation of emission controls at the MCC Plant has likely reduced emissions. Historical vegetation data suggest that fluoride transferred into plant tissues either through uptake from contaminated soils or through foliar absorption of hydrofluoric acid (HF) (Miller 1985, 1986, 1987). HF should be easily removed in the new stack scrubbing process, and the high level of fluoride in the underflow solids suggests that this process is now occurring. Fluoride in the underflow solids is likely in the form of fluorite, which is transported offsite via wind-borne transport. Particle deposition onto plants and soil surfaces appears to be the major current transport mechanism at the MCC Site. Figure 3-1 is an illustrative conceptual site model based on current RI data.

Organisms potentially most exposed in the affected areas (as delineated in Section 2.0) are soil organisms and burrowing small mammals (field mice, voles, ground squirrels and rabbits) to the north and south of the Plant. The coyote and badger are considered occasional feeders, while deer are considered infrequent transient foragers in the affected area. Also, cattle and horses grazing southwest of the Plant are considered to be potentially exposed to COPCs in soils, Soda Creek water, and in spring water. Songbirds and gamebirds may also be exposed to COPCs in soils; however, most of the pasture and agriculturally disturbed areas where COPCs exist do not provide attractive habitat to these birds.

The only potential exposure to threatened or endangered species (or candidates thereof), or sensitive species (Section 1.4.3) might be to the boreal owl, perigrine falcon or bald eagle. Exposure to these animals would presumably be via food chain transfer (primarily from rodents exposed to contaminated soils), and will be evaluated in subsequent portions of this section. The long-billed curlew, the goshawk and trumpeter swan are all transient, but do not prefer habitat found in contaminated areas associated with the MCC Plant. The hoary willow is reported only around Ledger springs, and green needlegrass is not expected to be found in agriculturally disturbed areas where contaminated soils exist.

3.2 EVALUATION OF STRESS

The estimated area of soils containing concentrations of COPCs greater than background is approximately 15 acres which generally border the perimeter of the Plant. The most highly affected areas are located along the north and south boundaries. Contaminant elevations in soils result from stack emissions, wind erosion from onsite source piles, and fugitive dusts from materials handling and roads (Golder Associates 1992).

The voluntary crop reduction field (planted in native grasses) along the north property line is thickly vegetated with a variety of grasses such as cheatgrass, fescue, bromegrass, and slender wheatgrass. During a field reconnaissance in June 1992, there was no visual evidence of plant stress (e.g., leaf discolorations or thinning or bare patches in the vegetative stand). Near the north fence line, the vegetation was somewhat dusty. Blowing dust was observed emanating

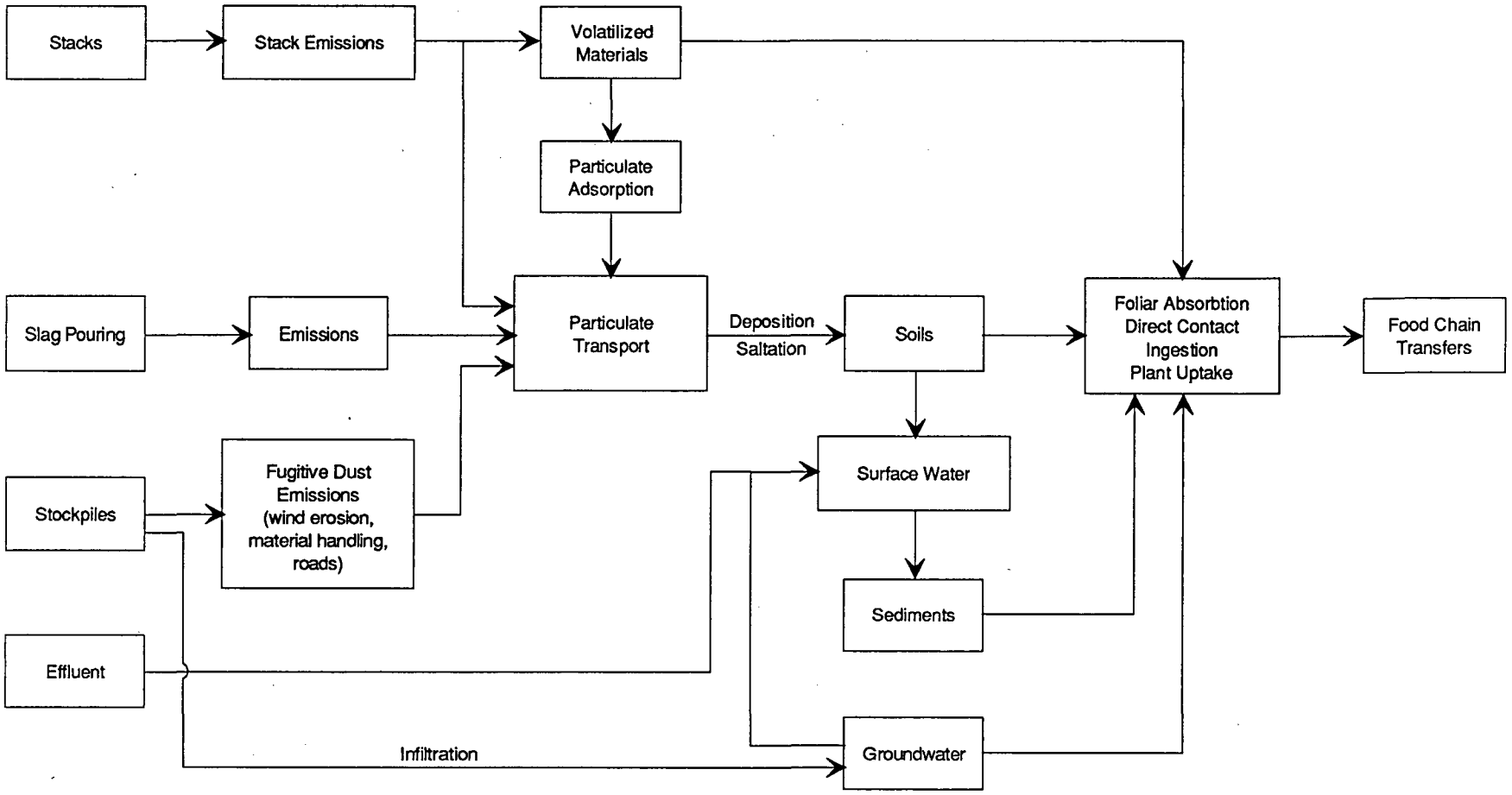

Figure 3-1

CONCEPTUAL SITE MODEL - CONTAMINANT MIGRATION TO ECOLOGICAL RECEPTORS
MONSANTO

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sporadically from some of the underflow solids and baghouse dust stockpiles. Saltation effects along portions of the source materials near the north fence line were noticeable, but were not noticeable outside the facility boundaries.

Vegetative stress in the horse and cattle pastures south and southwest of the MCC Plant, particularly in the Soda Creek watershed, was visible due to the effects of grazing. A few areas along Soda Creek were heavily grazed at the time of the field reconnaissance. It is expected that the effects of grazing and agricultural use on plant growth would mask noticeable effects from contaminants in the soil in these locations.

3.2.1 Highly Localized Receptors

Soils - In the evaluation of risk to these receptors, maximum concentrations of COPCs will be compared to plant phytotoxicity values (Sections 4.0 and 5.0). This comparison is used as an indication of potentially highly localized effects to the plant community, as well as an evaluation of effects on the producer-level ecosystem component.

Maximum concentrations represent a very conservative estimate of exposure concentrations. Soil concentrations likely represent contamination from current as well as historic deposition. Stack emissions have been reduced with the installation of scrubbers in the late 1980's. Particulate transport to off-site locations, mainly from the stacks and underflow solids pile, have been modelled and deposition rates calculated (Senes 1993). Current concentrations are assumed to represent future concentrations. Fate and transport of soil contaminants is detailed in the RI.

Sediments and Water - Similar to plants in soils, many benthic aquatic organisms are relatively immobile. Certain pelagic aquatic organisms, depending on the physical structure of their habitat may also be functionally non-mobile. As a conservative approach, maximum COPC concentrations in the two aquatic media will be compared to applicable sediment and water benchmark criteria (Sections 4.0 and 5.0). Chemical data will be combined with sediment toxicity tests and historical data to determine potential impacts to the aquatic ecosystem.

Again, maximum concentrations represent a conservative estimate of exposure concentrations. Sediment concentrations likely represent contamination from current as well as historic deposition. Contaminants may originate from soil runoff, but are more likely from ground water transport (Golder 1993). High concentrations of selenium and cadmium in ground water are mirrored by similarly high concentrations in sediments from immediately below the outfall to just south of the Mormon Spring/Creek watershed. Specific sediment concentrations may reflect depositional environments, which can be purged in episodic flood events; eventually, much of the sediments will deposit into Alexander Reservoir. Current concentrations are expected to vary spatially within the area of the Mormon Creek watershed, but in the absence of improved ground water quality, overall sediment concentrations are not expected to decrease. Maximum sediment concentrations are, therefore, considered to be reasonably conservative. Considerable uncertainty remains under potential future scenarios where periodic sediment transport could occur. However, future concentrations under these fate/transport assumptions and conditions cannot be accurately modelled.

Currently, the contaminated ground water plume characterized by Monsanto (Golder 1993) is migrating from the Plant toward the south and the plume may eventually emerge into areas along

the lower portions of Soda Creek. Given this assumption, it would be likely that sediment and water quality would be impacted. Due to the relatively high mobility of selenium and fluoride, these chemicals are likely to be COPCs under this scenario.

The extent of water and sediment contamination and toxicity would be influenced by a myriad of factors such as dilution in mixing zones, spatial distribution of depositional zones, redox potential of sediments, water hardness, and chemical complexation.

3.2.2 Moderately Mobile Receptors

Moderately mobile receptors are primarily exposed to COPCs through direct ingestion of soil, and to a lesser extent through limited food chain transfers. Field mice, voles, ground squirrels, and rabbits potentially fall into this category. Because no direct measurements of contaminant ingestion have been made, a conservative intake model has been developed to predict exposure to these organisms (Section 3.3) using the field mouse as a surrogate receptor. Mean concentration values for this category of receptors (Section 2.1.2) are used as input into these exposure models. Current exposure concentrations are assumed to represent future concentrations.

3.2.3 Transient Receptors

Similar to the moderately mobile receptors, direct ingestion of soils and food chain transfers account for the majority of exposure to transient receptors. Mule deer and coyote are examples of receptors in this category. The data summary developed for transient receptors (Section 2.1.2) will be input into the intake model discussed below. The horse pasture southwest of the Plant contains one soil sample, at the extreme edge of the pasture, with three radionuclides at approximately three times background. Risk to horses from these radionuclides may be evaluated if risks are found in more concentrated areas for other receptors.

Water ingestion by transient receptors will be evaluated in a separate intake model. Maximum concentrations from a pooled springs/creek data set will be used as input into the water intake model.

3.3 EXPOSURE MODELLING

The inadvertent ingestion of contaminated soils is considered a major intake route of contaminants in herbivores. Omnivorous and carnivorous animals can ingest soil particles trapped in body fur of their prey. Contaminants trapped in hair are also subject to ingestion during grooming processes. In addition, ingestion of contaminated water is an important exposure pathway.

An intake model has been developed to assess the level of exposure of COPCs in soils to ground dwelling small mammals and transient receptors using field mice and mule deer as surrogate receptors, respectively. Data regarding body weight and food intake levels, derived from animal research and veterinary diagnostics, have been used to estimate specific intake levels for food and water.

Field mice and mule deer were chosen to represent exposed wildlife species because of their likely presence in contaminated areas, their relative levels of exposure (due to differential ingestion patterns), and the availability of supporting literature for comparison.

3.3.1 Soil Ingestion

Table 3-1 summarizes intake assumptions used to model ingestion of soil. The mouse's diet is assumed to consist of three percent soil, and the deer's diet, two percent soil (Beyer and Conner 1991). Concentrations of COPCs in vegetation are assumed to be a function of soil concentration; these values are derived from plant/soil concentration ratios obtained through literature review. Total COPC intake by the field mouse and mule deer are calculated using the following equations:

$$Total\ intake = \frac{C_f * I_f + C_s * I_s}{BW}$$

$$C_f = BCV * C_s$$

where:

Total Intake	=	(mg/kg/d)
C_f and C_s	=	COPC concentrations in food and soil, respectively (mg/kg)
I_f and I_s	=	Intakes of food and soil, respectively (kg/d)
BW	=	Body weight of receptor (kg)
BCV	=	Bioconcentration Value (Unitless)

Table 3-1
Intake Assumptions for Soil Ingestion[a]

Species Parameter	Body Weight (kg)	Diet (kg/d)	Food (kg/d)	Soil Ingestion (kg/d)	Water Ingestion (l/d)
Mule Deer	60	1.50	1.47	0.03	6.0
Mouse	0.03	0.01	0.0097	0.0003	NA

a = Assumptions as modified from SAIC 1991, 1993.
NA = Receptor parameter not used

Total contaminant intake through soil ingestion is expressed in mg/kg of body weight per day (mg/kg/d). These units were chosen as a standard, although other units are commonly used. For example, some toxicity reference values (TRVs) are expressed in parts per million (ppm)

of feed. Feed concentration units assume a proportionality of body weight to feed intake and take into account species-specific rates of feed assimilation. Using these same assumptions, conversions were made units of ppm to units used in the model (mg/kg/d).

Intake calculations based on the model assumptions are presented in Appendix B (Tables B-1 and B-2). Plant/soil bioconcentration values (BCVs) have been derived from a range of values from the scientific literature. In the absence of literature values, BCVs are assumed, very conservatively, to be five percent. The absorbed fraction of chemical-specific intake doses are not widely available in the literature for the surrogate receptors. In addition, where absorption rates are reported, the levels vary considerably. Thus, it was conservatively assumed that the intakes were full bioavailable to the organism. Intake levels are compared with effect levels (TRVs) in Section 5.0.

3.3.2 Water Ingestion

Table 3-1 also lists the assumptions used to calculate water intake for the mule deer. COPC intake through water ingestion is calculated using the following equation.

$$Total\ intake = \frac{C_w * I_w}{BW}$$

where:

Total Intake =(mg/kg/d)
Cw = Concentrations of COPCs in water (mg/kg)
Iw = Intake of water (kg/d)
BW = Body weight of receptor

* Note that 1 L of water = 1 kg

Intake calculations based on water intake assumptions are presented in Appendix B (Table B-3). Intake levels are compared to effect levels in Section 5.

3.3.3 Food Chain Transfers to Higher Trophic Levels

In the areas of concern surrounding the MCC Plant, potential food chain transfers to carnivorous trophic levels is limited. Rodents, which are most exposed to soil contaminants, are a likely prey base for raptors. The habitat range of all of these birds, however, is much broader than the narrow habitat range of impacted rodents. Effective residence time and exposure is therefore very low. In addition, none of the COPCs identified in Section 2.0 have any substantial biomagnification properties. Overall food chain transfer is likely characterized by a rapid diffusion of chemical concentrations into a relatively small proportion of the ecosystem's higher order trophic levels. Although effects may occur at lower intake concentrations in the higher trophic level organisms, the high degree of contaminant diffusion suggests that this pathway is likely to be negligible. For example, a single transitory bald eagle may hunt rodents over an area of many thousands of acres. Rodents found in the roughly fifteen acres of contaminated soils adjacent to the MCC Plant would likely make up a negligible portion of the eagles diet.

Thus, the probability of exposure is considered negligible. This is also expected to hold true for the peregrine falcon and boreal owl. Therefore, these species will not be examined further.

3.4 EXPOSURE UNCERTAINTY

The uncertainty in exposure characterization is derived primarily from modelling assumptions and data summaries used for modelling input. Animal intake modelling encompasses several assumptions including soil, food, and water intake, as well as body weight and residence time. Reasonable ranges of these parameters are researched in the literature, and conservative ends of these ranges are selected as modelling assumptions. These assumptions are considered conservative, in that they tend to maximize exposure; true exposure is likely equal to or less than the modelled exposure. Dynamic interactions generally are not well characterized for ecosystems and become a major source of uncertainty. However, conservative characterization of habitat and extent of contamination may help to mitigate uncertainty in favor of the protection of ecological receptors.

4.0 CHARACTERIZATION OF ECOLOGICAL EFFECTS

This section summarizes available information concerning potential toxicological effects resulting from exposure to COPCs. The primary mechanism for evaluating toxicity is through analysis of quantitative dose-response relationships documented in the scientific literature. Ecological-effect endpoints are chosen to represent sensitive and/or indicative components of the ecosystem which may be impacted by chemical stressors. Sensitive and/or indicative indicators will presumably gauge the larger endpoint of detrimental effects to high level ecological organization (populations, communities, ecosystems). Section 5 will then combine this quantitative characterization of effects with the estimates of exposure (Section 3) to quantitatively estimate the risk of adverse effects, primarily in the localized ecosystem. Corollary qualitative endpoints may be used in a weight of evidence approach, in support of quantitative endpoints.

4.1 ECOLOGICAL ENDPOINTS

The introduction of contaminants into an ecosystem may cause environmental stress. Changes in birth rates, mortality, immigration, and emigration influence population sizes in an affected area. These changes also can cause shifts in productivity and spatial distribution of populations in a community. Such population-level effects determine the nature of alterations in community structure and function, such as variation in species diversity, simplification of food webs, and shifts in competitive advantages among species sharing limited resources.

To assist in evaluating potential stress to the localized ecosystem near the perimeter of the Monsanto facility, the following specific ecological endpoints or indicators are used to assess potential effects:

- Impairment of plant growth - Determined by comparison of soil chemical concentrations to literature derived toxicity reference values; correlated with visual inspection to provide indications of phytotoxicity from chemical contamination.

- Impairment of animal physiological processes - Determined by comparing literature based toxic reference concentrations to modelled intake concentrations to surrogate receptors at the individual level.

- Impairment of aquatic habitat - Determined by comparing sediment and water quality to proposed or promulgated criteria. Qualitative information (i.e., measured sediment or water toxicity) is also used.

- Deviation in structure and function compared to an unimpaired community - Qualitatively determined by comparing potentially affected areas with non-affected areas.

The remainder of this section is organized into two general areas. Section 4.2 presents a toxicity assessment based on chemical properties other than radioactivity. Toxic chemical effects are evaluated quantitatively.

Insufficient literature data are available to quantitatively characterize low-level radiation effects from specific radionuclides to ecological receptors; therefore, Section 4.5 will present a qualitative review of radiation effects.

4.2 TOXICOLOGICAL EFFECTS FROM COPCs

The media of concern for ecological effects resulting from operations at the Monsanto Plant are soils, Soda Creek water, spring waters, and sediments. Contaminants of potential concern were listed in Table 2-6. Concern arises as to whether the concentrations of these inorganic chemicals and radionuclides in the soils are potentially toxic to plants and/or animals.

Among the biological communities in the terrestrial environment, plants and soil invertebrates are likely to experience the greatest exposure to COPCs in soils. Limited data exist for the development of chemical-specific Toxicity Reference Values (TRVs) in soil. However, some TRVs for plants and soil invertebrates can be derived from numerous studies in the literature. The effects concentrations selected for this assessment are considered protective of most plant species relevant to this site. Table 4-1 presents literature-based TRVs capable of inducing toxic effects on plants (phytotoxicity).

In order to evaluate potential adverse effects to receptors such as small and large mammals, available toxicological data from the literature have been reviewed to select endpoints that would likely reflect the chosen exposure scenarios. Quantitative dose response information, representing chronic TRVs by the no-observed-adverse-effects-level (NOAEL) or the lowest-observed-adverse-effects-level (LOAEL) were selected where available for the field mouse surrogate.

More acute endpoints (e.g., LD_{50}), were selected from a variety of endpoints in the literature to represent acute TRVs. Because the mule deer is considered to be transient, acute endpoints were deemed acceptable, although some chronic endpoints were used because of literature availability. These chronic endpoints were not adjusted because they are already overly-conservative. Other endpoint selection criteria include giving preference to those endpoints having potential ecosystem-wide repercussions (e.g., reproduction).

The TRVs for chronic effects to field mice and mule deer are provided in Tables 4-2 and 4-3, respectively. The endpoints were selected to represent the types of effects in the representative organism. Surrogate receptors and endpoint types are typically used to make approximations due to a lack of specific literature data. For example, a search for an acute effect from cadmium on a mule deer might only yield data on chronic effects to cattle. Adjustment factors to account for interspecies differences may also compound uncertainties. Chronic effects may be used to screen for acute effects (more conservative). Much uncertainty remains in the endpoints, as well as in site specific data used in comparison. This uncertainty enables only rough approximations of risk when comparing exposure concentrations with TRVs. However, in this risk assessment, exposure assumptions are sufficiently conservative to mitigate any uncertainty which would underestimate receptor-specific toxicity. It should be noted that a safety

Table 4-1
Phytotoxicity Values for Chemicals of Potential Concern
in Soils

COPC	Potentially Phytotoxic Concentration[a] (mg/kg)	Endpoints
Arsenic	25	Endpoints are a compilation of numerous literature-derived endpoint effects. Effects often included reduction in growth, reduction in yield in grain crops and in vegetables. Most effects used to produce composite endpoint concentrations were chronic, low level physiological effects. Values represent a conservative summary of various endpoints evaluated. None were indirect physical effects.
Cadmium	10	
Chromium	100	
Fluoride	NA	
Selenium	30	
Silver	4	
Vanadium	100	
Zinc	250	
Radionuclides		
Lead-210	NA	
Polonium-210	NA	
Radium-226	NA	
Thorium-230	NA	
Uranium-238	NA	

NA = Data Not Available

a = Derived from the following sources: Kabata-Pendias, A and Pendias, H. (1991), ICF, Inc (1989), Adriano, D.C. (1986), Antonovics, J., et. al (1971), Chaney, R.L. (1980), CH2M Hill (1986a, 1986b), Davis, R.D. et. al (1978), Demayo, A. et. al (1982), Eisler, R. (1985 - 1988), Tyler, et. al (1989), Balsberg - Pahlsson (1989), Gorsuch et al. (1990).

Table 4-2
Chronic Toxicological Reference Values for a Field Mouse Exposed to Soil

COPC in Soil	Concentration (mg/kg/d)	Endpoint	Reference
Arsenic	20	Fetal/maternal mortality in mouse	CESARS 1992
Beryllium	0.54	NOAEL, rat	IRIS 1994
Cadmium	1.7	Depressed myocardial activity	CESARS 1992
Chromium III	1468	NOAEL[a]	IRIS 1994
Fluoride (soluble)	333	Fluoride induced changes in bone	NAS 1974
Selenium	0.45	Decreased birth weight, reproductive failure in rats	CESARS 1992
Silver	NA	NA	NA
Vanadium	15	Developmental toxicity - NOEL	SANCHEZ et al
Zinc	168 (1.68[c])	LD50 in mouse[b]	SAIC 1991
Radionuclides			
Lead-210	NA		
Polonium-210	NA		
Radium-226	NA		
Thorium-230	NA		
Uranium-238	NA		

NA = Data not available.
a = Based on Cr III which is the likely valence found in soil.
b = Best endpoint available; potential false negative conclusion.
c = A safety factor of 100 was applied in an effort to compensate for the acute endpoint.

Table 4-3
Toxicological Reference Values for Mule Deer Exposed to Soil and Water

COPC in Soil or Water	Concentration (mg/kg/d)	Endpoint	Reference
Arsenic	5.4	"No effect dose", Horse	Puls, 1989
Beryllium	0.54	NOAEL, Rat	IRIS 1994
Cadmium	66	Anemia, stillbirths; cattle	Puls, 1989
Chloride (water)	1000 mg/l	"Maximum tolerated water concentration" ; cattle	Puls 1989
Chromium	30	Scouring, dehydration, dermatitis (based on Cr VI)	Puls 1989
Copper	6.3	Decreased weight gain, low blood hemoglobin in pigs	CESARS 1993
Fluoride	19.8	"Highly toxic" in cattle (appears to refer to soluble Fluoride)	USEPA 1985
Fluoride (water)	2.5 mg/l	"Maximum recommended concentration"	Puls 1989
Nickel	49.5	Reduced feeding, cattle	Puls 1989
Nitrate/Nitrite	NA	NA	NA
Selenium	0.4	Reduced weight gain, cattle	Puls 1989
Silver	NA	NA	NA
Sulfate	NA	NA	NA
Vanadium	10	Diarrhea, immobility in cattle	Puls 1989
Zinc	29.7	Decreased weight gain in cattle	USEPA 1985
Radionuclides			
Lead-210	NA		
Polonium-210	NA		
Radium-226	NA		
Thorium-230	NA		
Uranium-238	NA		

NA = Data not available
a = Chromium III toxicity thresholds are not well known; at this site, trivalent chromium may be 95% of chromium found. Lower threshold refers to more toxic chromium VI.

factor has been applied to the zinc TRV for the field mouse because a chronic endpoint was not available. The zinc endpoint represents an acute response. A chronic or subchronic response may lie at a lower concentration.

Fluoride endpoints are based on soluble forms of fluoride; environmental forms may have differences in bioavailability. Insoluble fluoride may become somewhat more soluble under physiological conditions. It appears that most of the fluoride contamination associated with the Site is in the form of CaF_2. This is the result of very high calcium concentrations in the soil and very low solubility of this compound. The Site conceptual model suggests that most of the fluoride, which originally left the stack as HF, is now captured in the stack scrubbing process, and is limed and sent to the underflow solids as CaF_2. The solubility of fluoride is so low (< 16 mg/L), that transport into vegetation is minimal. Similarly, the increased solubility of an acid environment (i.e. the gut) is minimal; hence, bioavailability from all pathways is low, even when total fluoride concentrations may be high.

Available water and sediment quality criteria, designed to be protective of aquatic biota, are presented in Tables 4-4 and 4-5, respectively. Indicators of general toxicity in sediments include bacterial enzyme activity reduction, as well as algal growth reduction. Historical studies also have tested for lethal concentration effects (LC_{50} in fathead minnows) in effluent water. Visual observation for signs of stress can be used in aquatic media, as well as other media (i.e., soils) to assess general toxicity.

4.3 BIOLOGICAL TOXICITY TESTS

Monsanto collected sediment samples for biological toxicity tests in July of 1993. The results indicated the biological toxicity of sediments may be greater downstream of the outfall than upstream. Inherent toxicity in upstream control samples was observed. Downstream samples showed greater toxicity, relative to the control samples. Downstream samples were located at 100-feet and 2,400-feet from the outfall (SCSS-100 and SCSS-2400, respectively). The tests conducted included "Bacteria Enzyme Activity Reduction" as a function of sediment concentration, and "Algal Growth Reduction", also as a function of sediment concentration. SCSS-100 sediments were more toxic in terms of algal growth; SCSS-2400 sediment was more toxic in terms of bacterial enzyme activity. Toxicity under both tests was observed at the control location, but was less than both sampling locations under both tests. Results are presented in Table 4-6.

4.4 HISTORICAL DATA

Historical information relating to recorded or potential impacts to the area ecosystem have indicated negligible effects. A study by the State of Idaho regarding the MCC effluent line (Perry, 1976) indicates "a minor increase in nitrate, a decrease in phosphorous, and an increase in temperature in Soda Creek water". A Monsanto report from 1980 (Grothe, 1980) found no mortality on a Fathead Minnow (*Pimephales promelas*) from a 96 hour exposure to undiluted effluent water. Prior to the installation of stack emission controls, a group of cattle suffered from fluoride poisoning. This fluorosis may have been caused by exposure to contaminated

Table 4-4
Toxicity Reference Values for Water
and Water Quality Criteria
(mg/l)

COPC in Water	Concentration (mg/l)	Endpoint[a]	Reference
Cadmium[a]	0.004	Chronic Federal Water Quality Criteria	IRIS 1994
Nickel[a]	0.62	Chronic Federal Water Quality Criteria	IRIS 1994
Selenium	0.005	Chronic Federal Water Quality Criteria	IRIS 1994
Zinc[a]	0.42	Chronic Federal Water Quality Criteria	IRIS 1994
Fluoride	NA		
Chloride	NA		
Sulfate	NA		
Nitrate / Nitrite	High	Criteria not recommended because effect levels high.	IRIS 1994

a = Based on a hardness of 502.5 mg/l.

Table 4-5
Toxicological Reference Values for Sediments
Sediment Quality Criteria (SQC)
(mg/kg)

COPCs in Sediments	Concentration (mg/kg)	Endpoint	Reference
Arsenic	3 (10[a])	Great Lakes Harbor Sediments Guidelines (GLHSG)	USEPA 1977
Cadmium	1 (20[a])	Wisconsin Interior Criteria for Disposal of Sediments (WICDS)	WDNR 1985
Copper	25	GLSHG	USEPA 1977
Nickel	20 (70[a])	GLSHG	USEPA 1977
Selenium	1	WICDS	WDNR 1985
Silver	NA	NA	
Vanadium	NA	NA	
Polonium-210	NA	NA	

a = Adjusted SQC to accommodate high background concentrations.
NA = Data not available.

Table 4-6
Results of Soda Creek Sediment Biological Toxicity Test

Sample	Bacteria Enzyme Activity NOEC (%)	Algal Growth Reduction % of Control[a]
Upstream		
Control A	12.5	3
Control B	25	42
Control C	25	44
Mean	21	30
Downstream		
100 A	25	5
100 B	12.5	9
100 C	6.25	2
Mean	15	5
2400 A	6.25	10
2400 B	<6.25	15
2400 C	12.5	20
Mean	7.3	15

NOEC = No observed effects concentration
a = Algal Growth reduction reported at sample strength of 100%.

vegetation and groundwater. Apparently, high levels of hydrofluoric acid (HF) were released historically from the stacks. HF easily sorbs into vegetation which can be transferred to cattle through ingestion. Even with heavy burdens of CaF_2 in soils (from particulate deposition), biotransfer into plants is very low (personal communication, G. Miller, January 1994).

4.5 TOXICITY FROM EXPOSURE TO RADIATION

Detailed understanding and ultimate prediction of the distributions of radionuclides in the environment are difficult to achieve due to the complexity of ecosystems; the behavioral, morphological, and physiological intricacies of organisms; and, the chemical and physical dissimilarities among the radionuclides of concern.

Despite substantial research, the ability to pinpoint demonstrable ecological change to specific levels of radionuclide contamination is poor. Part of the problem is that the radiation dose to biological tissues from environmentally dispersed contaminants is highly variable, owing to the physical and chemical behavior of the material within a complex system. Another problem relates to quantifying subtle biological effects in complex systems at comparatively low radiation exposures. Large, fixed radiation sources have been used to irradiate ecosystems *in situ* at levels sufficient to produce observable effects; however, this research is only partially relevant to lower radiation levels from dispersed contaminants.

Noticeable effects at the population, community, and ecosystem levels require rather high doses of gamma radiation. Even though subcellular and cellular lesions may occur at low doses, they are not manifest in the measurable attributes of populations or communities. A few abnormal cells or organisms will likely not survive to perpetuate abnormality. Healthy, unaffected organisms will quickly fill voids or spaces made available through loss of affected individuals, and thus will dominate. Only with doses sufficient to cause substantial reduction in natality, can dramatic changes in populations and communities be seen. This is also true for other non-biomagnifiable chemicals. Intermediate level doses may cause slight reduction in community productivity, or shifts in species composition to less sensitive species, by reducing normal growth rates of sensitive species without causing basic alterations in community structure (IAEA, 1976; Ashby, 1967).

Few data exist at this time regarding the effects of chronic, low-level radiation doses to nonhuman species. Some low dose effects such as genetic anomalies, birth defects, decreased fertility, cancer, or premature aging may be possible, and the probability of observing an effect is generally assumed to increase with dose. At chronic doses of 100 rads of low-LET radiation, we can observe an increase of approximately 100% in the incidence of various genetic anomalies. The same 100 rad dose in an acute situation represents a lower bound of observed effects. Acute whole body doses (300 to 600 rad) to mammals will adversely affect the hematopoietic system and likely result in death within two to four weeks.

4.6 TOXICITY UNCERTAINTY

Endpoint selection is one of the greatest sources of toxicological uncertainty in a risk assessment. Literature review seldom reveals an ideal set of endpoints to be applied to a specific assessment. Toxicological reference values are usually derived from laboratory studies, which can vary substantially when applied to field conditions. In addition, surrogate receptors must often be used to approximate receptors found in study locations. Gross assumptions need to be made regarding toxicity and assimilation factors when using surrogate organisms. Often, there is uncertainty on how an effect will propagate into a population, community, or ecosystem.

Due to a lack of basic information (as reflected in the sparse literature) regarding chemical- and species-specific toxicity, quantification of uncertainty becomes difficult, at best. Therefore, results obtained by using these TRVs as best utilized as qualitative indicators used in a weight-of-evidence approach.

Occasionally, the TRVs chosen may not be conservative (e.g., an acute endpoint selected in place of a chronic endpoint, when chronic exposure is likely). These selections are noted in the risk analysis, because false negative indications of risk may occur.

Specific uncertainty lies in the general lack of specific radionuclide toxicity data (from radiation effects) for most organisms evaluated. Such data would aid in the evaluation of the incremental risk increase to ecosystem receptors through individual organisms.

In addition, a great deal of uncertainty lies in the selection of sediment quality criteria as TRVs. These values are based on habitats which are likely to be much more sensitive thant those evaluated in this assessment. Because of the very conservative nature of these values, comparisons will provide only indications of potential risks.

5.0 RISK CHARACTERIZATION

5.1 GENERAL APPROACH

This risk characterization integrates information from the exposure and effects assessments to estimate the risk of adverse effects to exposed populations and communities in an ecosystem. For an adverse effect to occur, two conditions must be met: (1) the contaminant must be present in the environment at concentrations sufficient to exert an adverse effect, and (2) the organism must come in contact (exposure) with the contaminant.

In this section, exposure point concentrations are compared with TRVs to evaluate potential risks to modelled receptor organisms exposed to soils and surface water. When receptors are exposed to concentrations that begin to cause adverse effects, there will be risk not only to that organism, but potentially to populations and communities on a broader ecological scale. Perturbations, measured as chemical stressor effects on single organisms, are only considered indicators of potential stress at these larger ecological and spatial scales. Direct comparison of intake concentrations to TRVs, when possible, should provide an indication of the potential for these chemically induced disturbances.

Given the overall conservative assumptions regarding exposure calculations as well as endpoint effects, minor exceedance of TRVs should be interpreted as only slight to moderate risk. If the intake concentration is more than one order of magnitude greater than the TRV, then the risks may be substantial to the individual organism modelled. Substantial individual risks may be translated into population or community effects by evaluating the individuals role in higher level organization.

5.2 RISKS FROM CHEMICAL TOXICITY

5.2.1 Soils

Highly Localized Receptors - Table 5-1 compares the Site surface soil concentrations of COPCs with the literature based TRVs for plants (phytotoxicity). As can be noted from the table, none of the COPCs fall into the category of potentially high phytotoxicity, except for cadmium and zinc. Concentrations of arsenic, chromium, cadmium, selenium, silver and zinc all fall into the category of slight to moderate risk. Table 5-2 indicates that the most frequent exceedances of phytotoxic TRVs occur at locations S-1, S2-3, S-4, S-10, S2-11, S-13, S-14, and MS2-7. As can be seen in Figure 2-2, these samples are located along the southern and northern edges of the MCC Plant. Any apparent toxicity associated with these chemicals was not evident during visual inspection in the field (i.e., observable plant stress). However, endpoints associated with the phytotoxic TRVs may not be visually evident on a small scale. Phytotoxic effects on some plants may be inconspicuous or be masked by a competitive shift to more tolerant species. In addition, phytotoxic endpoints derived under laboratory control can vary dramatically in the field. Extrapolation of endpoints to other plant species is also a source of uncertainty.

Table 5-1
Comparison of Highly Localized Exposure Point Concentrations with Phytotoxic TRVs

Potential Contaminant	Maximum EPC	Phytotoxic TRV	Frequency EPC Exceeding TRV		
			Low < TRV	Moderate <10X TRV	High >10X TRV
(mg/kg)					
Arsenic	34	25	38/39	1/39	
Cadmium	168	10	23/41	14/41	4/41
Chromium	325	100	34/41	7/41	
Fluoride	136	NA	ND		
Selenium[a]	109	30	6/7	1/7	
Silver	13	4	35/41	6/41	
Zinc	2670	250	30/41	10/41	1/41
Radionuclides (pCi/g)					
Lead-210	65	NA	ND		
Polonium-210	77	NA	ND		
Ra-226	17	NA	ND		
Th-230	18	NA	ND		
Uranium-238	16	NA	ND		

a = limited data set
TRV = Toxicity Reference Value.
EPC = Exposure Point Concentration.
ND = Risk Not Determined because of data gap.
NA = TRV Not Available.

Table 5-2
Sample Locations where Concentrations Exceeded Phytotoxic TRVs

Location	10X TRV > conc.> TRV	conc.> 10X TRV
S-1	Ag, Zn, Cd	
S2-3	Cr, Ag, Zn, Cd	
S-4	Cr, Cd, Zn	
S-8	Cd	
S-9	Cd, Zn	
S-10	Cr, Ag, Zn, Cd	
S2-11	Cr, Ag	Cd, Zn
S-12	Cd, Zn	
S-13	As, Cr, Ag, Zn, Se	Cd
S-14	Cr, Ag, Zn	Cd
S-15	Cd	
MS2-2	Cd	
MS2-3	Cd	
MS2-7	Cr, Zn	Cd
MS2-8	Cd, Zn	
MS2-11	Cd	
MS2-30	Cd	
MS2-35	Cd	

It appears unlikely that structural and/or functional properties have been significantly altered within the plant community. This conclusion is based on the relatively small affected area (the area restricted to the periphery of the Plant), the low to moderate potential for adverse effects on plants, and the lack of obvious damage (visually) from chemical contamination to plant biota surrounding the Plant.

Moderately Mobile Receptors - Table 5-3 compares mouse intake concentrations to TRVs. All intake concentrations, except cadmium and selenium, are less than TRVs, indicating slight to moderate risks from exposure to these chemicals in the soil. Large uncertainty lies in the endpoints chosen for zinc and the characterization of selenium in the soils, as discussed in Section 4.0. Food chain transfers to other trophic levels are also likely to be minimal, given the relatively low biomagnification of any of these metals in animals. Risks from specific radionuclides are generally unknown. Carcinogenic risks are likely to be low because of the short lived species; reproductive effects may be greater due to relatively high fecundity.

Transient Receptors - Table 5-4 indicates low risk to deer from exposure to soils around the MCC Plant. All intake concentrations are less than TRVs. As with the mouse, uncertainty lies primarily in choosing an endpoint that may not be sufficiently conservative. In addition, only

Table 5-3
Comparison of Mouse Intake Concentrations with Chronic TRVs

Potential Contaminant	Intake Concentration	Mouse TRV (mg/kg/d)	Intake Concentration Exceeding TRV		
			Low< TRV	Moderate < 10 X TRV	High > 10 X TRV
(mg/kg/d)					
Arsenic	0.7	20	•		
Cadmium	3.2	1.7		•	
Chromium	3.1	1,468	•		
Fluoride	2.6	333	•		
Selenium	1.6	0.45		•	
Silver	0.1	NA	ND		
Vanadium	5.3	15	•		
Zinc	58.2	168	•		
Radionuclides (pCi/g/d)					
Lead-210	1.3	NA	ND		
Polonium-210	1.2	NA	ND		
Radium-226	0.4	NA	ND		
Thorium-230	0.3	NA	ND		
Uranium-238	0.2	NA	ND		

NA = TRV Not Available.
ND = Risk Not Determined due to a lack of toxicity data.
TRV = Toxicity Reference Value (mg/kg/d) or (pCi/g).

Table 5-4
Comparison of Mule Deer Intake Concentrations with Chronic TRVs

Potential Contaminant	Intake Concentration	Deer TRV	Intake Concentrations Exceeding TRV		
			Low < TRV	Moderate < 10 X TRV	High > 10 X TRV
Inorganics (mg/kg/d)					
Arsenic	0.01	5.4	•		
Cadmium	0.03	66	•		
Chromium	0.04	30	•		
Fluoride	0.03	19.8	•		
Selenium[a]	0.02	0.4	•		
Silver	0.004	NA	•		
Vanadium	0.07	10	•		
Zinc	0.2	29.7	•		
Radionuclides (pCi/g/d)					
Lead-210	0.01	NA	ND		
Polonium-210	0.01	NA	ND		
Radium-226	0.001	NA	ND		
Thorium-230	0.001	NA	ND		
Uranium-238	0.006	NA	ND		

a = limited data set
NA = TRV Not Available.
ND = Not Determined due to a lack of toxicity data.
TRV = Toxicity Reference Value (mg/kg/d).

toxic effects are evaluated; carcinogenic effects are not represented due to a lack of literature data. Carcinogenic effects from radionuclides are more likely in this group of receptors due to longer life; however, large uncertainty remains.

Extrapolation of surrogate results to other transient receptors, such as coyote, song/game birds and raptors may be done by employing a safety factor to the deer intake. The safety factor would be based on species sensitivity, differential exposure to soil, bioaccumulation, etc. If the safety factor were set at two orders of magnitude, selenium would become of moderate concern (extrapolate Table 5-4). If the safety factors were set at three orders of magnitude, arsenic, chromium, fluoride, vanadium and zinc would also be of moderate concern. When employing safety factors such as these, there is a great deal of uncertainty is generated. However, a safety factor of 1,000 is substantial, and does suggest that risks are probably minimal to this class of receptors, based on extrapolation from surrogate receptors.

5.2.2 Water and Sediments

Transient Receptors - Transient deer exposed to Soda Creek and Spring water will likely not suffer any chronic effects, with the possible exception of fluoride toxicity at Calf Springs and the Mormon Springs Complex. Table 5-5 compares intake concentrations to TRVs. All intake concentrations, except for fluoride, are less than TRVs, indicating generally low risk to the mule deer in the event of occasional exposure to these waters. Other transient receptors with higher residence times may experience moderate risk from fluoride at the Mormon Spring Complex. The TRVs and model parameters represent a large source of uncertainty.

Sediment and Water Quality - Table 5-6 compares sediment and water COPC concentrations to available criteria or reference concentrations. Cadmium and selenium exceed acute and chronic water quality criteria (WQC) in the springs only. These exceedances are reflective of ground water entering the surface water. The habitat at these springs is not generally considered "aquatic" habitat but rather a discharge into a developing stream. Because cadmium and selenium levels have not been measured in Soda Creek south of the Mormon Springs complex, potential impacts to the water are unknown. However, it is expected that fate and transport mechanisms for these two chemicals would result in rapid removal from the water column followed by deposition into the sediments.

Arsenic, cadmium, copper, nickel, and selenium all exceed their respective sediment quality reference levels. These exceedances also appear to be linked to ground water entering the Mormon/Soda Creek watershed southwest of the Plant. Data regarding fate and transport mechanisms associated with contaminant deposition into sediments and sediment transport in the Soda Creek watershed are unavailable. Results of biological toxicity tests in sediments also seem to correlate with exceedances in Soda Creek, from the outfall line to 2,400 feet downstream of the outfall. Sediment toxicity, however, does not automatically translate into risk for the aquatic receptors. The "natural" environment is very harsh in the upper reaches of Soda Creek, due mainly to highly carbonated water. If a naturally rich benthic community with multiple trophic levels existed in upper Soda Creek, it would be likely that the measured toxicity (as indicated by biological tests and quantitative exceedance over TRVs) would result in higher potential for adverse impacts. The risks appear to be low however, because there is a very limited habitat to be impacted.

Table 5-5
Comparison of Mule Deer Water Intake Concentrations with TRVs (mg/kg/d)

Potential Contaminant (All from springs except as noted)	Intake Conc.	TRV	Intake Concentration Exceeding TRV		
			Low < TRV	Moderate < 10 X TRV	High > 10 X TRV
Cadmium	0.002	66	•		
Chloride (Soda Creek)	15.9 (159 mg/l)	1000 mg/l	•		
Sulfate	22	NA	ND		
Fluoride	0.3 (3 mg/l)	2.5 mg/l		•	
Nitrate	5.2	NA	ND		
Nickel	0.003	49.5	•		
Selenium	0.019	0.4	•		
Zinc	0.015	29.7	•		

Number in parenthesis indicates converted values to match TRV endpoint units.
NA = TRV Not Available.
ND = Risk Not Determined because TRV unavailable.
TRV = Toxicity Reference Value (mg/kg/d).

Table 5-6
Comparison of Water and Sediment COPC Concentrations to Water and Sediment TRVs

COPC	Concentration	TRV	Media Concentration Exceeding TRV		
			Low < TRV	Moderate < 10 X TRV	High > 10X TRV
WATER	mg/l	mg/l			
Cadmium	0.018	0.004			•
Nickel	0.03	0.62	•		
Selenium	0.19	0.005 (0.020)			•
Zinc	0.151	0.42	•		
Fluoride	3	NA	ND		
Chloride	159	NA	ND		
Sulfate	220	NA	ND		
Nitrate/Nitrite[a]	5.2	High	•		
SEDIMENTS	mg/kg	mg/kg			
Arsenic	49.3	10		•	
Cadmium	61	20		•	
Copper	42.4	25		•	
Nickel	153	70		•	
Selenium	347	1			•
Silver	1.3	NA	ND		
Vanadium	208	NA	ND		
Polonium-210 (pCi/g)	3.3	NA	ND		

Value in parenthesis indicates acute water quality criteria.
TRV = Toxicity Reference Value
a = Measurement assumed to represent nitrate.

Based on limited ground water modelling as part of the RI (Golder 1993), the ground water contaminant plume south of the MCC Plant is anticipated to discharge into the mid to lower reaches of Soda Creek. Such discharge may result in increased contamination of the sediments and water. In the event that the plume intercepts more sensitive reaches of lower Soda Creek, or even Alexander Reservoir, measurements of media COPC concentrations would be necessary to evaluate potential impacts to ecological receptors. Speculation on media concentrations without empirical data is subject to enormous uncertainty. A myriad of factors could potentially influence diluting or concentrating mechanisms. Reducing environments in sediments can immobilize aqueous selenium into high concentrations into those sediments. Subsequent oxidation can mobilize high selenium concentrations for transport to ecological receptors. Dilution in mixing waters may also play a substantial role in reducing COPC concentrations, which may also aid in reducing sediment loads. The natural hardness of the water which receives the plume may substantially affect the toxicity of many potential COPCs.

In addition, pulse events such as storms, may mobilize years worth of currently accumulated sediments and deposit them into the next downstream sediment sink. Ultimately, some of the sediments may deposit into Alexander Reservoir.

5.3 RISKS FROM RADIATION TOXICITY

Adverse ecological effects from gamma radiation near the MCC Plant are negligible. Overall, gamma radiation does not exceed background for the soil samples. Gamma radiation measurement serves as a general indicator of elevations of gamma-emitting radioactive chemicals in soils. The resolution of this method, however, precludes detection of slight radionuclide elevations measurable using specific radionuclide analysis. In addition, this method does not detect elevations of alpha or beta emitting radionuclides. In the human health risk assessment, elevated radionuclides have shown incremental risk above background for individual humans.

For non-human receptors, this type of quantification of individual organism risk is not feasible. In addition, when assessing risks to ecological organization above the individual, the slight incremental risk to the individual becomes of less concern. From Appendix E of the Human Health Risk Assessment, an exposure model may be applied to calculate the dose to ecological receptors. Based on the UCL background gamma reading of 19.8 μR/hr, dose equivalents to receptors would be equal to 0.02 mRem/hr, or roughly 0.5 mRem day. According to the IAEA (1992), 100 mRem/day would not cause population-level effects, even to the most sensitive terrestrial species.

Elevations in gamma-radiation may, therefore, serve as a better environmental indicator of potential for ecosystem impacts than measurements of specific radionuclides at levels detected at this site.

5.4 SUMMARY OF RISK

The comparison of site exposure concentrations to toxicological reference values presented in Sections 5.2 and 5.3 suggests that plant and animal receptors near the Site (as represented by

modelled surrogates) would be at low to moderate risk from contaminants in the various media. The distribution of COPCs is not widespread in soils, but is concentrated along the north and south periphery of the Plant boundary. Areas of water and sediment contamination are also relatively localized. However, a variety of wildlife species exist at these locations. Fluoride in Mormon Spring may pose a risk to sensitive transient receptors. COPCs in sediments may also pose risks to aquatic organisms. Qualitative indicators have not been observed which indicate these risks are being manifested. Based on field observations, the habitat surrounding the MCC Plant and associated wildlife usage do not appear to have been altered when compared to similar habitat conditions in other portions of the valley.

Risks may be extrapolated to domestic livestock which may be impacted by contaminated media. An example is the watering of livestock from the (b) (6) well. A qualitative review of data from the last three sampling rounds indicate concentrations of all COPCs (except zinc) are noticeably below those found in Mormon Spring. At Mormon Spring, fluoride was of concern to sensitive transitory species. At the (b) (6) well, fluoride concentrations were approximately ten times less than Mormon Spring, thus should not be of concern. Elevations of zinc at the (b) (6) well do not indicate risk to modelled receptors. No other domestic animals are known to be exposed to contaminated ground water associated with the MCC Plant.

5.5 RISK CHARACTERIZATION UNCERTAINTIES

Uncertainties associated with this risk assessment include: the use of conservative assumptions with regard to exposure parameters for the modeled receptors; the selection of TRVs for comparison with predicted intakes; and, extrapolation of overall potential ecological risks from an evaluation of a few selected receptor organisms representative of local biota.

Fate and transport mechanisms (e.g. sediment transport to more sensitive habitats), as well as data gaps, such as selenium in Soda Creek water, also provide additional sources of uncertainty. Because contaminated ground water from the plume south of the plant is currently affecting the surface water and sediments of Mormon and Soda Creeks, it is likely that additional loading of COPCs, such as selenium, will continue into the future. This additional loading into the sediments may increase, especially in portions of the mid-to-lower Soda Creek watershed as sediment are transported downstream, and result in increased risks to environmental receptors. Also, uncertainty exists regarding the ground water plume migrating south of the Plant. Future impacts from potential transport mechanisms of COPCs are difficult to predict, due to uncertainties of nature and extent of future contamination.

6.0 SUMMARY AND CONCLUSIONS

The ecological risk assessment was generally conducted using a weight of evidence approach. Evidence considered included qualitative information gathered during Site reconnaissance, as well as quantitative comparisons used in the risk analysis. Based on these factors and the results of Site characterization, the assessment considered the following exposure scenarios:

- Potential exposures to threatened, endangered or sensitive speices;
- Vegetation exposed to potential phytotoxic levels of chemicals in soil;
- Ingestion of potential contaminants in soils by field mice and mule deer;
- Ingestion of potential contaminants in spring water by deer; and,
- Exposure to aquatic communities of potential contaminants in sediments and surface water.

Based on field observations, the habitat surrounding the MCC Plant does not appear to have been altered when compared to similar habitat conditions in other portions of the valley. The findings of the ecological risk assessment support the following conclusions, as related to the assessment endpoints defined in Section 1.0:

- Critical, sensitive, or unique habitats have not been impacted by releases from the MCC Plant. This conclusion was made primarily in Section 2.0, when areas of contamination were determined to not contain any of these habitats. Habitats distributions were described in Section 1.0.
- Impacts to threatened or endangered species are considered negligible. This conclusion was made primarily in Section 3.0 when it was determined that: food chain transfers are negligible to the boreal owl, bald eagle and perigrine falcon; the trumpeter swan, goshawk, long-billed curlew, hoary willow and green needlegrass do not prefer habitat associated with contaminated media around the Plant during any important phase of their life-cycles.
- Some risks may occur to specific organisms associated with the localized ecosystem around the Plant, although the structure and function of this ecosystem does not appear to have been altered (conclusion drawn in Section 5.0). Specific conclusions regarding these potential risks include:
  - Sensitive plant species near the northern Plant boundary may be at risk from cadmium and zinc in the soils;
  - Moderately mobile organisms (as modelled by field mice) may be at risk from cadmium and selenium, mainly north of the Plant boundary;

- Chronic exposure to other elevated metals in soil in field mice and deer would result in low toxicological risk (the predicted chemical intake exposures are less than the TRVs);

- Sensitive transient organisms (as modelled by the deer) may be at risk from fluoride concentrations in the Mormon Springs complex. Domestic stock watering (e.g. for cattle and horses) from the Mormon Springs complex may also pose a hazard due to fluoride;

- Sensitive aquatic organisms may be at risk from selenium and cadmium in surface water and sediments, particularly in Mormon creek and the portion of Soda Creek in the vicinity of Mormon creek.

In general, the comparison of Site exposure concentrations to toxicological reference values presented in Sections 5.2 and 5.3 suggests that plant and animal receptors near the Site (as represented by modelled surrogates) would not be at substantial risk from contaminants in soils. The distribution of COPCs is not widespread in soils but rather is concentrated around the perimeter of the Plant boundary. Potential for ecosystem risk from other media has not recently manifested; the mechanism of cattle fluorosis in the past appears to have been removed with installation of emission controls. However, spring water may still provide a pathway for fluoride effects. Sediment contamination and toxicity within portions of Mormon Creek and Soda Creek may also increase in the future (Golder 1993). In addition, if the modelled ground water plume emerges into mid to lower Soda Creek, potential impacts will be highly dependent on many factors, as discussed in Section 5.2.2.

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Appendix A

Vegetation and Wildlife Species in the Region

APPENDIX A
COMMON VEGETATION SPECIES BY COVER TYPE IN THE REGION

SAGEBRUSH-GRASS COVER TYPE

Common Name	Scientific Name
Shrubs:	
Antelope bitterbrush	*Purshia tridentata*
Big sagebrush	*Artemisia tridentata var. tridentata*
Black sagebrush	*Artemisia arbuscula var. nova*
Chokecherry	*Prunus virginiana*
Horsebrush	*Tetradymia spp.*
Mountain big sagebrush	*Artemisia tridentata var. vaseyana*
Oregon grape	*Berberis repens*
Rabbitbrush	*Chrysothamnus viscidiflorus*
Saskatoon serviceberry	*Amalanchier alnifolia*
Shrubby cinquefoil	*Potentilla fruticosa*
Silver sagebrush	*Artemisia cana*
Snowberry	*Symphoricarpos oreophilus*
Forbs:	
Arrowleaf balsamroot	*Balsamorhiza sagitatta*
Aster	*Aster spp.*
Eriogonum	*Eriogonum spp.*
Fleabone daisy	*Erigeron spp.*
Gland cinquefoil	*Potentilla fruticosa*
Horsemint	*Agastache urtricifolia*
One-flower helianthella	*Helianthella uniflora*
Penstemon	*Penstemon spp.*
Scarlet paintbrush	*Castilleja chromosa*
Showy goldeneye	*Viguiera multiflora*
Sticky geranium	*Geranium viscosissimum*
Tapertip hawksbeard	*Crepis acuminata*
Tarragon sagebrush	*Artemisia dracunculoides*
Western yarrow	*Achillea millefolium*
Grasses:	
Big mountain brome	*Bromus carinatus*
Bluebunch wheatgrass	*Agropyron spicatum*
Cheatgrass	*Bromus tectorum*
Great Basin wildrye	*Elymus cinereus*
Idaho fescue	*Festuca idahoensis*
Medusa head	*Elymus caput-medusae*
Oniongrass	*Melica bulbosa*
Prairie junegrass	*Koeleria cristata*
Sandberg bluegrass	*Poa secunda*
Slender wheatgrass	*Agropyron trachycaulum*
Meadow Foxtail	*Alopecurus pratensis*
Green Needlegrass	*Stipa viridula*

APPENDIX A (Continued)
SELECTED TERRESTRIAL WILDLIFE SPECIES IN THE REGION

MAMMALS	
Common Name	Scientific Name
Weasels	*Mustela spp.*
Badger	*Taxidea taxus*
Striped skunk	*Mephitis mephitis*
Coyote	*Canis latrans*
Red fox	*Vulpes fulra*
Bobcat	*Lynx rufous*
Yellowbelly marmot	*Marmota flaviventris*
Ground squirrels	*Citellus spp.*
Chipmunks	*Eutamias spp.*
Northern pocket gopher	*Thomamys talpoides*
Deer mouse	*Peromyscus maniculatus*
Muskrat	*Onadatra zibethica*
Porcupine	*Erithizon dorsatum*
Snowshoe hare	*Lepus americanus*
Whitetail jackrabbit	*Lepus townsendi*
Mule deer	*Odocoileus hemionus*

BIRDS	
Common Name	Scientific Name
Red-tailed hawk	*Buteo jamaicensis*
Swainson's hawk	*Buteo swainsoni*
Goshawk	*Accipiter gentilis*
Golden eagle	*Aquila chrysuetos*
Peregrine falcon	*Falco peregrinus*
Prairie falcon	*Falco mexicanus*
Ruffed grouse	*Bonasa umbellus*
Sage grouse	*Centrocercus urophasianus*
Sharptailed grouse	*Pedioecetes phasianellus*
Boreal owl	*Aegolius funereus*
Great horned owl	*Buba virginianus*
Common nighthawk	*Chordeiles minor*
Marsh wren	*Cistothorus palustris*
Loggerhead shrike	*Lannus ludoricianus*
Yellow warbler	*Dendroica petechia*
Western meadowlark	*Sturnella neglecta*
White pelican	*Pelecanus erythrorhynchos*
Eared grebe	*Podiceps nigricollis*
American bittern	*Botaurus lentiginosus*
Northern harrier	*Circus cyaneus*
American kestrel	*Falco sparverius*
American coot	*Fulica americana*
Black-chinned hummingbird	*Archilochus alexandri*

APPENDIX A (Continued)
SELECTED TERRESTRIAL WILDLIFE SPECIES IN THE REGION

BIRDS

Common Name	Scientific Name
Horned lark	*Piranga ludoviciana*
Western Tanager	*Carpodacus cassinii*
Reef finch	*Pooecetes gramineus*
Vesper Sparrow	*Pica pica*
Black-billed magpie	*Corrus brachyrhnchos*
American crow	*Parus atricapillus*
Black-capped chickadee	*Cyanocitta stelleri*
Stellers Jay	*Turdus migritorius*
Robin	*Sialia mexicana*
Western bluebird	*Anascarolinensis*
Green-winged teal duck	*Anas platyrhynchas*
Mallard duck	*Aythya americana*
Redhead duck	*Anas acuta*
Northern Pintail	*Anas strepera*
Gadwall duck	*Ardea heroidias*
Great blue heron	*Grus canadensis*
Greater sandhill crane	*Cygnus buccinator*
Trumpeter swan	*Brawta canadensis*
Canada goose	*Numenius americanus*
Long-billed curlew	*Capella gallinago*
Common snipe	*Larus californicus*
California gull	*Larus pipixcan*
Franklin's gull	*Anas cyanoptera*
Cinnamon Teal	*Anas clypeata*
Northern Shoveler	*Hirundo rustica*
Barn swallow	*Anas americana*
American widgeon	*Aytha americana*

AMPHIBIANS

Common Name	Scientific Name
Tiger salamander	*Ambystama tigrenum*
Western toad	*Bufo boreas*
Leopard frog	*Rana pipiens*

REPTILES

Common Name	Scientific Name
Sagebrush lizard	*Sceloporus graciosus*
Gopher snake	*Pituophis melanoleucus*
Gartersnakes	*Thamnophis spp.*
Racer	*Coluber constrictor*

Appendix B

COPC Intake Calculations

Table B-1
COPC Intake Calculations for a Mouse within Localized Areas

COPC	Mean Soil Concentration (mg/kg)	BCV	Concentration In Vegetation (mg/kg)	Body Weight (kg)	Diet (kg/d)	Soil Ingestion (kg/d)	Food Ingestion (kg/d	Total COPC Intake (mg/kg/d)
Arsenic	26.1	0.05	1.31	0.03	0.01	0.0003	0.0097	0.7
Cadmium	122.4	0.05	6.1	0.03	0.01	0.0003	0.0097	3.2
Chromium	237.5	0.01	2.4	0.03	0.01	0.0003	0.0097	3.1
Fluoride	99.7	0.05	5.0	0.03	0.01	0.0003	0.0097	2.6
Selenium[a]	59.4	0.05	3.0	0.03	0.01	0.0003	0.0097	1.6
Silver	9.5	0.01	0.10	0.03	0.01	0.0003	0.0097	0.1
Vanadium	399.0	0.01	4.0	0.03	0.01	0.0003	0.0097	5.3
Zinc	2225.0	0.05	111.3	0.03	0.01	0.0003	0.0097	58.2
Radionuclides (pCi)								
Lead-210	50.3	0.05	2.51	0.03	0.01	0.0003	0.0097	1.3
Polonium-210	44.3	0.05	2.21	0.03	0.01	0.0003	0.0097	1.2
Radium-226	13.5	0.05	0.7	0.03	0.01	0.0003	0.0097	0.4
Thorium-230	11.9	0.05	0.61	0.03	0.01	0.0003	0.0097	0.3
Uranium-238	12.5	0.01	0.11	0.03	0.01	0.0003	0.0097	0.2

BCV = Bioconcentration Value = [veg]/[soil]
a = Exposure concentration derived from limited data set.

Table B-2
COPC Intake Calculations for a Deer in the Migration Area

COPC	Concentration (mg/kg)	BCV	Concentration In Vegetation (mg/kg)	Body Weight (kg)	Diet (kg/d)	Soil Ingestion (kg/d)	Food Ingestion (kg/d)	Total COPC Intake (mg/kg/d)
Arsenic	9.7	0.05	0.5	60	1.5	0.03	1.47	0.02
Cadmium	38.9	0.05	1.9	60	1.5	0.03	1.47	0.07
Chromium	89.5	0.01	0.9	60	1.5	0.03	1.47	0.07
Fluoride	29.5	0.05	1.5	60	1.5	0.03	1.47	0.05
Selenium	21.9	0.05	1.1	60	1.5	0.03	1.47	0.04
Silver	5.2	0.05	0.3	60	1.5	0.03	1.47	0.01
Vanadium	192.8	0.01	1.9	60	1.5	0.03	1.47	0.14
Zinc	562.1	0.01	5.6	60	1.5	0.03	1.47	0.14
Radionuclides (pCi)								
Lead-210	14.3	0.05	0.7	60	1.5	0.03	1.47	0.02
Polonium-210	12.6	0.05	0.6	60	1.5	0.03	1.47	0.02
Radium-226	7.9	0.05	0.4	60	1.5	0.03	1.47	0.01
Thorium-230	9.2	0.05	0.5	60	1.5	0.03	1.47	0.02
Uranium-238	7.0	0.05	0.4	60	1.5	0.03	1.47	0.01

BCV = Bioconcentration Value = [veg]/[soil]
a = Exposure concentration derived from limited data set.

Table B-3
COPC Intake Calculations for a Mule Deer
from Spring and Creek Waters

Chemical Spring Water Unless Noted	Maximum Concentration (mg/kg)	Body Weight (kg)	Water Ingestion (l/d)[a]	PC Ingestion (mg/kg/d)
Cadmium	0.018	60	6	0.002
Nickel	0.03	60	6	0.003
Selenium	0.19	60	6	0.019
Zinc	0.151	60	6	0.015
Fluoride	3	60	6	0.3
Chloride (Soda Creek)	159	60	6	15.9
Sulfate	220	60	6	22
Nitrate	5.2	60	6	0.52

a = l/d is assumed to equal kg/d.